Phytomedica

Course Notes

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Asian Concepts of Digestive Health

Ayurveda

The simple approach to digestive disease outlined by Samuel Thomson, developed further by the later physiomedicalists, is remarkably similar to the approaches used in Ayurveda. The "heat" of the stomach referred to by Thomson is more or less synonymous with the concept of *agni* in Ayurveda. Beyond the role assigned to it by Thomson, the concept of *agni* includes metabolic functions, and so also relates to specific organs such as thyroid, liver, skeletal muscles, and the skin. In its more subtle form, *agni* represents the mind's ability to "digest" sensory information, including our ability to perceive, comprehend, and discriminate between all the different facets of experience.

Qualities and characteristics of agni

Agni is characterized by the qualities of *ushna* ('hot'), *tikshna* ('sharp'), and *laghu* ('light'), extending outwards from in the *amashaya* (stomach and small intestine) as the *jatharagni*. Here the *jatharagni* attends to the separation of food into its 'subtle essence' (*sukshma rasa,* which feeds the mind), its 'gross nutrient' portion (*rasa*, which feeds the body), and 'waste products' (*kitta,* including both feces and urine).

According to Ayurveda, when a *dosha* is in an increased state, the qualities that each *dosha* manifests can have negative effects upon digestion:

- In *vattika* conditions the *jatharagni* is *vishamagni:* digestion that is erratic and irregular.
- In *paittika* conditions the *jatharagni* is *tikshnagni*: extremely intense, with a strong appetite, burning sensations, and thirst.
- In *kaphaja* conditions the *jatharagni* is *mandagni (agnimandya)*, characterized by weak digestion, poor appetite, nausea, and epigastric heaviness.

In the absence of *dosha* increase or vitiation, the *jatharagni* is *samagni*: correct, proper and normal.

Qualities and characteristics of ojas

Ojas is a bodily substance that counter-balances the activities and qualities of *agni.* There are two types of *ojas:* one called the *para ojas* (the 'superior essence'), and the called *apara ojas* (the 'inferior essence'). *Para ojas* is also referred to as the 'eight drops' *(ashtabindu)*, and is an unchanging substance that is intrinsic to the manifestation of life that only dissipates upon death. In contrast, *apara ojas* maintains the strength of the body and mind, and is in a continual state of flux, dependent upon factors such as proper breathing, a healthy diet, and good digestion.

Once the ingested food is processed by *agni* the nutrient portion *(rasa)* is circulated to each of the bodily tissues *(dhatus),* nourishing and supporting their various functions. Each bodily tissue also maintains its own subset of *agni*, and some portion of the received nourishment is further refined by this *agni* into *apara ojas*. Simply referred to as *ojas*, this fluctuating aspect of the vital essence plays an important role in supporting digestive function, and just as *ojas* is dependent upon *agni*, so does *ojas* sacrifice itself to nourish *agni.* Factors that enhance the status of *ojas* include rest, sleep, breath control *(pranayama),* and meditation, whereas *ojas* is depleted by poor digestion, over-thinking, strong emotions, insomnia, physical exercise, work, toxins, disease, and sexual activity.

The status of *ojas* can be assessed by the luster of the eyes, the strength of limbs, and the function of the mind and senses. The greatest concentration of *ojas* is found in the reproductive tissue, which uses this *ojas* to create life. In health *ojas* is for the most part distributed equally all over the body, or is directed to support specific functions, such as the senses or digestive organs, when they are active. In acute disease or trauma the flow of *ojas* is blocked, and in chronic disease the flow of *ojas* gradually becomes deficient. When diminished, the lack of *ojas* (called *ojakshaya)* produces symptoms such as fear, anxiety, weakness of the senses, poor complexion, poor memory, poor concentration, and emaciation, all of which correspond to an increase in *vāta.*

Qualities and characteristics of ama

According to Ayurveda, a primary factor in digestive illness is the accumulation of *ama,* or 'undigested food,' which primarily is the result of *mandagni.* As the by-product of poor digestion, *ama* is opposite in nature to *agni*, displaying qualities such as *guru* ('heavy'), *shita* ('cold'), *snigdha* ('greasy'), *sthira* ('stable'), *picchila* ('slimy'), and *manda* ('slow'). As *ama* accumulates, it counters the light, hot, and sharp qualities of *agni* and impairs digestion further. This leads to a diminishment of *ojas*, and because *ojas* is the substrate upon which *agni* itself is nourished, a vicious cycle is established, resulting in a progressive increase in *ama* and a commensurate decrease in both *agni* and *ojas. Ama* thus represents a state of digestive entropy, and its accumulation over a sustained period eventually robs *ojas* and *agni* of their functionality, and hastens the degenerative process.

As they share qualities in common there is some confusion between *ama* and *kapha,* and often they are mistakenly believed to be the same thing. *Kapha* represents an aspect of disordered

homeostasis as an **endogenous** mechanism that leads to the expression its characteristic qualities. In contrast, *ama* is an **exogenous** substance derived from weak digestion *(agnimandya),* and hence, typically arises from the influence of *kapha.* Once *ama* is generated, however, it can associate with any of the *doshas* to cause their increase and vitiation. In such a state a *dosha* is said to be *sama*, or 'with *ama',* whereas in the absence of *ama*, a *dosha* is said to be *nirama,* or 'without *ama'*. Sometimes the term *ama dosha* is used more generally to describe a state in which *ama* has accumulated and is causing an imbalance.

Although *ama* is not the only cause of disease in Ayurveda, it is the most frequent cause, and thus at the outset of treatment for almost any condition the elimination of *ama* and the enhancement of *agni* is implemented, in a process called *ama pachana ('ama* cooking'). Quite often simply by dispelling *ama* and restoring *agni* the condition will resolve, but if the persists beyond the use of *ama pachana*, a specific line of treatment is administered to the vitiated *dosha(s).*

The following table describes the differences between *sama* and *nirama* conditions:

***Sama* conditions**	***Nirama* conditions**
mucus congestion, catarrh	mucus normal, thin, clear
poor appetite	good appetite
indigestion, symptoms after eating	digestion occurs unnoticed
lethargy and lassitude after eating	energized and revitalized after eating
constipation	at least two bowel movements daily
sinking, dark-colored stools with mucus	floating, yellowish brown stools; no mucus
increased urination, urgency, frequency	normal urination
thick tongue coating	clear or thin white coating
headache	no headache
circulatory congestion, feeling of coldness,	circulation normal
numbness, tingling, neuralgia	nervous function normal
loss of strength	normal strength
joint swelling, pain, and inflammation	normal joint function
orbital edema, eyes appear dull	no orbital swelling, eyes bright/shining
symptoms worse with cold/damp weather/climates, worse at night; better with heat and dryness	health unaffected by changes in weather or climate

Table 3: Sama and nirama

In a broader context, the accumulation of *ama* is the impairment of one's ability to derive nourishment from life, be it physical, emotional, mental, or spiritual. A correctly functioning *agni* thus confers a harmonious benefit to the whole organism, with proper discrimination of the body, mind, and senses. It is important to remember that Ayurveda considers the partaking of food to be a *yagya,* or spiritual sacrifice. According to Ayurveda, *agni* is a sacrificial fire that resides within each of us, and when we consume food, our digestion becomes a catalyst that allows us to receive a great blessing of abundance. Proper digestion in Ayurveda thus depends upon the proper degree of mindfulness, as well as reverence, for this blessing obtained from the consumption of food.

- 96 g – guggulu (*Commiphora wightii,* purified oleo-gum resin)
  - Rx: 2 pills bid-tid

Traditional Chinese medicine

Digestive function in Traditional Chinese medicine is largely ruled by the Stomach *(wei)* and Spleen *(pi)*, which are responsible for generating the Food Essence *(gu qi)* that forms the basis of the Post-Natal Essence *(hou tian zhi qi).* Like *apara ojas* in Ayurveda, the Post-Natal Essence is responsible for sustaining the energy of the body. Traditional Chinese medical theory states that the Stomach is a *yang* organ, whereas the Spleen is a *yin* organ. Based on this dichotomy, *yin* is thus an important element to counter-balance the function of the Stomach, whereas *yang* is a vital component to counter-balance the Spleen.

Stomach

The function of the Stomach in Chinese medicine is to denature the ingested food, analogous to the way a compost pile breaks down plant material, only requiring moisture and heat in the process. After this rotting and ripening process the Stomach propels the ingested food downwards into the Small Intestine *(xiaochang),* which separates out the nutrient portion of the food. After this, the nutrient portion is the transported to the Spleen for further processing, and the wastes are sent to the Large Intestine *(dachang)* to be eliminated. Similar again to Ayurveda, the Stomach is the root of health in Chinese medicine, and the first step in transforming the ingested food into the energy that feeds the body. Thus if the Stomach is in a weakened state, the *qi* cannot be maintained and will eventually be lost.

The Stomach is particularly sensitive to any irregularities in diet, and if excessively cold foods are consumed, such as raw vegetables and cold water, this has the effect of weakening *yang*, promoting symptoms such as poor appetite, loose motions, and generalized coldness. Conversely, when excessively spicy or drying foods are consumed this can promote a deficiency of *yin* within the Stomach, with symptoms such as afternoon fever, thirst, and constipation. If excessively spicy and greasy foods are consumed the result may be excess heat within the Stomach, which similarly weakens the *yin* component, but leads to specific symptoms such as epigastric burning, thirst, constant hunger, nausea, and bad breath.

If the Stomach lacks the *qi* to properly ripen the ingested food, this manifests as a Stomach *qi* deficiency, leading to a general weakening of *qi* in the body, manifesting symptoms such as poor appetite, epigastric discomfort after eating, loose motions, and weakness of the limbs. This latter syndrome is often referred to as 'food stagnation', in which the ingested food cannot be ripened, leading to symptoms such as burping, epigastric heaviness, and poor appetite. When the Stomach *qi* is 'rebellious' and flows upwards instead of downwards, usually in association with excess Stomach heat, there may be symptoms of gastric reflux, burping, hiccoughs, and vomiting.

Based on the correct identification of these various patterns, herbs and formulas can be chosen to restore balance to the Stomach, including:

1. Stomach *yang* deficiency:
   - herbs, e.g. gan jiang (*Zingiber officinalis* rhizome), wu zhu yu (*Evodia rutaecarpa* fruit), chuan jiao (*Zanthoxylum bungeanum* pericarp), ding xiang (*Syzigium aromaticum* flower bud), gao liang jiang (*Alpinia officinarium* rhizome), bi ba (*Piper longum* fruit), hu jiao (*Piper nigrum* fruit)
   - Li Zhong Wan (Regulate Middle Pill)
     - 9 g – gan jiang (*Zingiber officinalis* rhizome)
     - 9 g – ren shen (*Panax ginseng* root)
     - 9 g – bai zhu (*Atractylodes macrocephala* root)
     - 9 g – zhi gan cao (*Glycyrrhiza uralensis* root, stir-fried in honey)
       - granules, Rx: 2-4 g bid
   - Wu Zhu Yu Tang (Evodia Decoction)
     - 9-12 g – wu zhu yu (*Evodia rutaecarpa* seed)
     - 18 g – gan jiang (*Zingiber officinalis* rhizome, recently dried)
     - 9 g – ren shen (*Panax ginseng* root)
     - 12 pieces – da zao (*Ziziphus jujuba* fruit)
       - decoction, Rx: 200 mL bid

2. Stomach *yin* deficiency
   - herbs, e.g. sa shen (*Adenophora verticillata* root), mai men dong (*Ophiopogon japonicus* root), shi hu (*Dendrobium nobile* stem), yu zhu (*Polygonatum odoratum* root)
   - Yi Wei Tang (Benefit Stomach Decoction)
     - 9 g – sha shen (*Adenophora verticillata* root)
     - 15 g – mai men dong (*Ophiopogon japonicus* root)
     - 15 g – sheng di huang (*Rehmannia glutinosa* root)
     - 4.5 g – chao yu zhu (*Polygonatum odoratum*, dry-fried root)
     - 3 g – bing tang (rock sugar)
       - Rx: 200 mL bid-tid
   - Mai Men Dong Tang (Ophiopogon Decoction)
     - 15-64 g – mai men dong (*Ophiopogon japonicus* root)
     - 9 g – ren shen (*Panax ginseng* root)
     - 6-15 g – jing mi (non-glutinous rice)
     - 12 piece – da zao (*Ziziphus jujuba* fruit)
     - 6 g – gan cao (*Glycyrrhiza uralensis* root)
     - 6-9 g – zhi ban xia (*Pinellia ternata* rhizome, fried with ginger, vinegar, or alum)
       - decoction, Rx: 200 mL qid
       - granules, Rx: 2-4 g bid-tid

3. Stomach Fire
   - herbs, e.g. ge gen (*Pueraria lobata* root), sheng di huang (*Rehmannia glutinosa* root), zhi mu (*Anemarrhena asphodeloides* rhizome), gua lou ren (*Trichosanthes kirilowii* seed), huang lian (*Coptis chinensis* rhizome), tian hua fen (*Trichosanthes kirilowii* root)
   - Ban Xia Xie Xin Tang (Pinellia Decoction to Drain the Epigastrum)
     - 9-12 g – zhi ban xia (*Pinellia ternata* rhizome, fried with ginger, vinegar, or alum)
     - 9 g – gan jiang (*Zingiber officinalis* rhizome, recently dried)

    - 9 g – huang qian *(Scutellaria baicalensis* root)
    - 3 g – huang lian *(Coptis chinensis* rhizome)
    - 9 g – ren shen *(Panax ginseng* root)
    - 12 pieces – da zao *(Ziziphus jujuba* fruit)
    - 9 g – zhi gan cao *(Glycyrrhiza uralensis* root, stir-fried in honey)
        - harmonizes Stomach, directs Rebellious *qi* downwards, disperses distension
        - granules, Rx: 3-4 g bid-tid
- Mai Men Dong Tang (Ophiopogon Decoction)
    - decoction, Rx: 200 mL qid
    - granules, Rx: 2-4 g bid-tid

4. Stomach *qi* deficiency (Food Stagnation)
    - herbs, e.g. shan zha *(Crataegus pinnatifida* fruit), mai ya *(Hordeum vulgare,* fermented sprout), gu ya *(Oryza sativa* fermented sprout), lai fu zi *(Raphanus sativus* seed), chen pi *(Citrus reticulata* peel), pei lan *(Eupatorium fortunei* herb), cao dou kou *(Alpinia katsumadai* seed)
    - Bao He Wan (Preserve Harmony Pill)
        - 9-15 g – shan zha *(Crataegus pinnatifida* fruit)
        - 9-12 g – shen qu (massa fermentata, medicated leaven)
        - 6-9 g – lai fu zi *(Raphanus sativus* seed)
        - 6-9 g – chen pi *(Citrus reticulata* peel)
        - 9-12 g – zhi ban xia *(Pinellia ternata* rhizome, fried with ginger, vinegar, or alum)
        - 9-12 g – fu ling *(Poria cocos* fruiting body)
        - 3-6 g – lian qiao *(Forsythia supensa* seed)
            - tea pills, Rx: 5-8 pills bid-tid
            - granules, Rx: 3-5 g bid-tid
    - Xiang Sha *yang* Wei Pian (Saussurea Amomum Nourish Stomach Pill)
        - 1.5 g – ren shen *(Panax ginseng* root) or dang shen *(Codonopsis pilosula)*
        - 3 g – bai zhu *(Atractylodes macrocephala* rhizome)
        - 2.4 g – fu ling *(Poria cocos* fruiting body)
        - 2.4 g – cang zhu *(Atractylodes lancea* rhizome)
        - 2.4 g – jiang zhi chao hou po *(Magnolia officinalis*, fried with ginger)
        - 2.4 g – chen pi *(Citrus reticulata* peel)
        - 2.4 g – chao xiang fu *(Cyperus rotundus*, dry-fried rhizome)
        - 2.1 g – bai dou kou *(Amomum kravanh* seed)
        - 1.5 g – mu xiang *(Saussurea costus* root)
        - 2.4 g – sha ren *(Amomum villosum* seed)
        - 3 g – *Angelica sinensis*
        - 1.5-3 g – zhi gan cao *(Glycyrrhiza uralensis* root, stir-fried in honey)
        - 1.5-3 g – da zao *(Ziziphus jujuba* fruit)
            - tablets, Rx: 3-5 tabs bid-tid
            - granules, Rx: 2-4 g bid-tid

5. Rebellious Stomach *qi*
    - herbs, e.g. sha ren (*Amomum villosum* seed), mu xiang *(Saussurea costus* root), ding xiang *(Syzygium aromaticum* flower buds)

Gastroesophageal reflux disease

Gastroesophageal reflux disease (GERD) is a somewhat newly recognized pathology that describes a condition previously known as "heartburn." GERD is characterized by a burning sensation behind the sternum, often accompanied by regurgitation of the stomach contents into the mouth or lungs. GERD may also manifest as respiratory symptoms such as cough, wheezing, and hoarseness, and may be confused with other conditions such as chronic bronchitis or asthma. Patients with GERD may also present with diminished salivary secretion and an increased risk of tooth decay and gum recession.

The mechanism of GERD is attributed to a dysfunction of the **lower esophageal sphincter (LES)** that lies between the stomach and esophagus. The LES is composed of smooth muscle, and normally only relaxes upon swallowing. With delayed stomach emptying and an increase in gastric pressure, however, the acidified chyme fails to properly clear the esophagus, blocking the function and eventually weakening the integrity of the LES. The acid itself causes damage to the esophageal epithelium, which lacks the alkaline mucous secreted in the stomach, causing esophageal erosion and ulceration. Chronic inflammation may result in the accumulation of scar tissue and a stricture (narrowing) of the esophagus, and there may be a replacement of the normal squamous epithelium with abnormal **columnar (Barrett's) epithelium,** which is considered to be precancerous.

Most episodes of GERD occur during the day, usually after eating, although some sufferers will also experience reflux during sleep. The nocturnal form of GERD is associated with a higher risk of esophagitis because during sleep the patient produces less saliva and swallows less often, which is required clear out and neutralize the acid. Factors that weaken the integrity of the LES include smoking, caffeine, chocolate, fatty foods, overeating, tight clothing, a hiatal hernia, and certain medications are all associated risk factors for GERD.

Medical Treatment

The medical treatment of GERD consists of lifestyle modifications, drug treatments, and surgery. The patient is encouraged to be aware of which foods or activities tend to make the problem worse, such as smoking, or the consumption of caffeinated foods and beverages, chocolate, and fatty foods. Tight clothing around the torso increases intra-abdominal pressure is to be avoided. Body position is also considered to be an important aspect in managing GERD, and recommendations might be made to maintain an upright posture after eating, ensuring that the ingested food does not reflux back into the esophagus. Some patients may be counselled to insert a wedge under their back at night to keep the esophagus above the stomach while sleeping. Similarly, patients are counselled to avoid exertion after a meal, such as bending or lifting, as this contracts the abdominal muscles and forces food back up through the weakened LES. Patients are also recommended to eat in a relaxed manner, and eat smaller meals. Patients that are obese are often a greater risk of GERD because of the excess abdominal fat that puts pressure on the stomach. Similarly, pregnant women often complain of heartburn, simply because of the pressure placed upon the stomach from the growing fetus, but also because hormonal fluctuations tend to make the esophageal and gastric mucosa more sensitive and therefore more reactive.

Gastric impairment leading to GERD can also be found in diabetic patients suffering from gastroparesis, as well as in neurological disorders such as Parkinson's disease. In some cases gastric impairment is a symptom associated with system pathologies such as scleroderma in which the dysfunction of the LES is attributed to autoimmune-induced fibrosis. Commonly used oral medications linked to GERD and gastric disease, include acetyl salicylic acid (ASA) and ibuprofen, which are directly toxic to the gastric mucosa, and well as potassium supplements, and the antibiotic tetracycline that often promote burning sensations in the esophagus.

The classical medical approach to GERD relies use of over-the-counter (OTC) antacid medications such as calcium carbonate that neutralize stomach acid. Although recommended for only occasional use many patients are encouraged or end up using them on a chronic basis, which has a negative effect upon gastric secretion and weakens stomach function. Another similar regimen is the use of bismuth subsalicylate that acts to coat the lining of the stomach and suppress acid secretion. Like other salicylates, however, it is likely that bismuth subsalicylate is also toxic to the gastric mucosa.

Prescription medications include promotility agents, histamine H2-receptor antagonists (H2 blockers), and proton pump inhibitors (PPIs). Promotility drugs such as the drug cisapride, metoclopramide and bethanechol are used to promote gastric motility. Cisapride in particular has since been recognized to have some dangerous effects including ventricular tachycardia and ventricular fibrillation, as well as diarrhea, gastric pain, headaches, and constipation. More commonly, H2 blockers (e.g. cimetidine, famotidine, nizatidine, ranitidine) and PPIs (e.g. omeprazole, lansoprazole, rabeprazole and pantoprazole) are prescribed to reduce the amount of acid produced in the stomach. The theory behind the use of H2 inhibitors and PPIs is that an excess secretion of stomach acid underlies GERD, even though most patients with this condition usually have lower than normal gastric acid levels. Although the use of acid-suppressing agents can give the esophageal epithelium time to heal, they often promote the underlying issue because they weaken gastric function further.

PPIs are among the top-selling drugs in the world, despite the fact that they come with some serious risks. Used long term PPIs can increase the risk of *Clostridium difficile* infection, pneumonia, and interfere with mineral absorption such as magnesium, promoting the risk of muscle spasm, heart palpitation, and convulsion. The general diminishment in nutrient absorption when taking PPIs increases the risk of bone fractures, and also inhibits the production of intrinsic factor, impairing the absorption of vitamin B12 and increasing the risk of pernicious anemia. More recently, the use of PPIs has been linked to an increased risk of dementia, kidney disease, and heart attack.[41]

Surgery is an alternative to prescription drugs when treatment is unsuccessful, or when certain complications arise. Negative effects of surgery can occur in up to 20% of patients, such as difficulty swallowing or the inability to belch or vomit. Frequently the benefits are limited, and patients will often require the continued use antacid drugs post-surgery to control symptoms.

[41] Goodman B. *Research Evaluates Possible Link to PPI Risks*. WebMd. June 8, 2016. Available from: http://www.webmd.com/heartburn-gerd/news/20160608/proton-pump-inhibitor-health-risks

Holistic Treatment

From a Western herbal perspective GERD is viewed as an upper GI tract digestive deficiency. Impaired secretion and poor motility leads to a commensurate weakening of the LES, causing the gastric contents to reflux back into the esophagus. Thus to resolve GERD treatment must be directed towards restoring gastric secretion and motility. Nonetheless, temporary measures are often required to neutralize the refluxed acids and promote healing of the esophagus. Generally this is best undertaken by eating small meals of starchy foods with only small amounts of protein, and very little fat (see the graduated diet, p. 182). Flour products, however, which have a glue-like consistency and impair gastric motility should be avoided. Likewise, the basic principles of food combining should also be followed, such as avoiding the consumption of animal proteins with carbohydrates, or eating fruit after meals.

In Ayurveda, GERD is resembles a condition called *amlapitta* ('sour bile'), which describes the reflux of stomach acid *(amla)* into the esophagus commensurate with an impairment in biliary excretion *(pitta).* The underlying factor, however, relates to *mandagni* or weak digestion. Likewise in Chinese medicine, most cases of GERD are caused by Cold and Damp affecting the Spleen, along with Liver *qi* stagnation, causing Stomach Fire and Stomach *yin* deficiency. In both Ayurveda and Chinese medicine the goal is to clear the heat, restore gastric digestion, and promote healing. At the outset of treatment this can be achieved by using bitter-tasting herbs that have a mild laxative effective. Bitter-taste in particular has a number of interesting effects on its own, promoting the secretion of gastrin by stimulating chemoreceptors on the tongue. This in turn stimulates the secretion of gastric juices, the closure of the LES, and the opening of the ileo-cecal sphincter, promoting proper motility. Bitter herbs also appear to modulate the secretion of cholecystokinin (CCK), which allows for proper gastric churning and the secretion of bile and pancreatic juices in anticipation of the chyme moving into the duodenum. In Ayurveda, however, too much of the bitter taste tends to inhibit digestion, so bitters need to be used in judicious doses, and are often combined with carminatives and stimulants to offset this effect. Likewise in both Chinese medicine and Ayurveda, herbs with a sweet flavour are used to reduce heat and promote healing, as well as carminative and digestive stimulants to restore proper function to the stomach

The following is a review of the holistic strategy used to address GERD:

Reduce esophageal inflammation

- demulcents: shatavari *(Asparagus racemosa* root), mai men dong *(Ophiopogon japonicus* root), tian men dong *(Asparagus cochinchinensis* root), slippery elm *(Ulmus fulva* inner bark), marshmallow *(Althaea officinalis* root), comfrey *(Symphytum officinalis* leaf/root)
    - prepared as cold infusion or as powders taken with honey
- de-glycyrrhized licorice (DGL): 2–3 tablets, chewed ad lib
- fresh aloe *(Aloe vera* leaf juice): 15-25 mL bid-qid
- banana, as ripened fruit, banana chips, or leaf powder, ad lib
- alkaline remedies: shankha bhasma *(Turbinella pyrum* calcinated shell ash), shukti bhasma *(Ostrea gigas* calcinated shell ash), pravala bhasma (calcinated coral ash)

- laxative botanicals to purge excess heat, used for a short period during the initial stages of treatment, e.g. turkey rhubarb *(Rheum palmatum* root), cascara sagrada *(Rhamnus purshianus* wood), trivrit *(Operculina turpethum* root)
  - use with carminatives e.g. ginger *(Zingiber officinalis* rhizome), fennel *(Foeniculum vulgare* seed), ajwain *(Trachyspermum ammi* seed)
- astringents, to promote muscular tone of the LES, check bleeding and heal ulcerations, e.g. cranesbill geranium *(Geranium maculatum* root), oak *(Quercus alba* bark), avens *(Geum urbanum* leaf), goldenseal *(Hydrastis canadensis* root/rhizome), bayberry *(Myrica cerifera* bark), goldenrod *(Solidago canadensis* herb), fir *(Abies grandis* bark)
- flavonoids and flavonoid-containing botanicals to limit acid production via inhibiting histamine release, e.g. nettle *(Urtica dioica* leaf), calendula *(Calendula officinalis* flower), amalaki *(Phyllanthus emblica* fruit), meadowsweet *(Spiraea ulmaria* herb), chamomile *(Matricaria chamomilla* flower), huang qian *(Scutellaria baicalensis* root), chai hu *(Bupleurum falcatum* root), green tea extract, quercitin

Promote proper gastric digestion and motility

- bitters, taken in small doses before meals, e.g. barberry *(Berberis vulgaris* root), gentian *(Gentiana lutea* root), centaury *(Erythraea centaurium* root), buckbean *(Menyanthes trifoliata* root), goldenseal (*Hydrastis canadensis* root/rhizome)
  - use of bitters are often avoided at the outset of treatment because the initial stimulation of acid production may worsen any esophageal ulceration
- carminatives and stimulants, to counter the effects of bitter-tasting herbs, and to enhance digestion, e.g. calamus (*Acorus calamus* rhizome), fennel (*Foeniculum vulgare* seed), chamomile *(Matricaria chamomilla* flower), aniseed *(Pimpinella anisum* seed)
  - use caution with pungent botanicals such as ginger and cayenne, and "upward moving" aromatics such as mint *(Mentha* spp.), caraway (*Carum carvi* seed), lavender *(Lavandula angustifolia* flower)
- digestive enzymes, full spectrum (i.e. HCl, pancreatic enzymes, ox bile): 2 – 3 caps with meals
  - used primarily in the older patients; ideally only short term
- dietary factors
  - food combining: avoid mixing animal proteins with starchy food, fruit should only be consumed on an empty stomach
  - avoid dairy and flour products, which due to their sticky and heavy properties impair gastric motility
  - avoid overeating
  - avoid eating within three hours of bedtime
  - avoid alcohol
  - avoid tobacco
  - avoid deep-fried foods, e.g. French fries, potato chips, etc.
- weight loss, to reduce intra-abdominal pressure

Promote healing of epithelium

- avoid Factors that promote intestinal permeability, p. 166

- vulneraries, e.g. calendula *(Calendula officinalis* flower), plantain *(Plantago spp.* leaf), selfheal *(Prunella vulgaris* leaf), St. John's wort *(Hypericum perforatum* flower), licorice *(Glycyrrhiza glabra* root), chickweed *(Stellaria media* herb)
- bone broth
- nutrients
  - vitamin A: 25,000-50,000 IU daily
  - vitamin C: 1 – 2 g bid – tid, to bowel tolerance
  - vitamin E: 800-1200 IU daily
  - zinc citrate: 50 mg daily
  - methylsulfonylmethane (MSM): 2 – 3 g, bid – tid

Formulations - Ayurveda

- Dhanyapanchakam churna
  - as a general digestive aid with balanced effects
  - 1-2 grams given with warm water twice daily
- Avipattikara churna, 1-2 grams given with warm water twice daily
  - for *pitta* symptoms
  - 1-2 grams given with warm water twice daily

Formulations – Chinese medicine

- Ban Xia Xie Xin Tang (Pinellia Decoction to Drain the Epigastrum)
  - harmonizes Stomach, directs Rebellious *qi* downwards, disperses distension
  - granules, Rx: 3-4 g bid-tid
- Mai Men Dong Tang (Ophiopogon Decoction)
  - benefits Stomach, nourishes *yin,* descends *qi*
  - decoction, Rx: 200 mL qid
  - granules, Rx: 2-4 g bid-tid
- Xiao Yao San (Rambling Powder)
  - spreads Liver *qi,* strengthens Spleen
  - powder, Rx: 6-9 g bid-tid with 6 g wei jiang *(Zingiber officinalis*, baked rhizome) and 3 g of bo he *(Mentha haplocalyx* herb)
  - granules, Rx: 2-4 g bid-tid

Formulations – Unani

- Jawarish-E-Kamooni
  - 70 g – zeera siyah *(Carum carvi* seed)
  - 70 g – barg-e-sudab *(Ruta graveolens* leaf)
  - 70 g – filfil siyah *(Piper nigrum* fruit)
  - 70 g – zanjabeel *(Zingiber officinale* rhizome)
  - 20 g – bura-e-armani (silicates of alumina and iron oxide)
  - 1 kg – qand safaid (sugar)
    - used for *humuzat-e-meda* (hyperacidity), *fuwaq* (hiccough), *nafkh-e-shikam* (flatulence in the stomach), qabz (constipation)
    - confection, Rx: 10-15 g bid-tid

- 9-15 g – wei rou dou kou *(Myristica fragrans*, roasted seed)
- 6-15 g – he zi *(Terminalia chebula* fruit)
- 6-20 g – mi zhi *ying* su ke *(Papaver somniferum* pericarp)
- 9-15 g – bai shao *(Paeonia lactiflora* root)
- 6-12 g – dang gui *(Angelica sinensis* root)
- 6-9 g – mu xiang *(Saussurea lappa* root)
- 6-9 g – zhi gan gao *(Glycyrrhiza uralensis* root, stir-fried in honey)
  - warms Spleen, restores deficiency, stops diarrhea
  - Rx: 200 mL bid-tid

- Si Shen Wan (Four Miracle Pill)
  - 4 parts – bu gu zhi *(Psoralea corylifolia* seed)
  - 1 part – chao wu zhu yu *(Evodia rutaecarpa* dry-fried seed)
  - 2 parts – rou dou kou *(Myristica fragrans* seed)
  - 2 parts – wu wei zi *(Schisandra chinensis* fruit)
    - warms Spleen, binds up intestines, stops diarrhea
    - Rx: 200 mL bid-tid

Formulations – Unani

- Habb-E-Raal
  - pill (500 mg), Rx: 1-2 pills bid, after meals

Formulations – Western herbal

- Dr. Christopher's Diarrhea Formula
  - 6 parts – bistort *(polygonum bistorta* root)
  - 6 parts – raspberry *(Rubus idaeus* leaf)
  - 1 part – Composition Powder
    - decoction, 30-90 mL very few hours

Small intestine bacterial overgrowth (SIBO)

Small intestine bacterial overgrowth (SIBO) is a digestive disorder caused by the infiltration and accumulation of colonic bacteria within the small intestine. Normally the small intestine is relatively sterile, as the digestive secretions of the stomach and small intestine exert a powerful antimicrobial effect to limit bacterial growth. As the chyme passes from the small intestine to the colon through the ileocecal sphincter it is inoculated by bacteria within the cecum, after which colonic bacteria ferment the chyme to form the feces. In SIBO, however, there is a retrograde flow of bacteria into the small intestine through the ileocecal sphincter, where they take up residence and interfere with the process of digestion and absorption. Excessive bacterial concentrations in the small intestine cause direct inflammation to the epithelial cells leading to diarrhea, whereas improperly digested nutrients such lipids, proteins, and carbohydrates that pass into the colon induce diarrhea by osmosis.

Patients with SIBO typically develop signs and symptoms associated with **irritable bowel syndrome (IBS),** including nausea, bloating, vomiting, diarrhea and/or constipation, malnutrition, weight loss, and malabsorption. Considered to some extent a "waste-basket"

diagnosis, recent research has demonstrated that up to 78% of IBS patients likely suffer from SIBO.[42] Some patients may lose weight and children with SIBO may fail to thrive. Impaired fat digestion evidenced by steatorrhea may also occur, leading to deficiencies of fat-soluble nutrients and vitamins. Likewise, SIBO may impair the absorption of iron and vitamin B12, and lead to chronic anemia.

There are a number of factors that cause or promote SIBO, including immunosuppression from the use of immunosuppressant medications, as well as both acquired and inherited immunodeficiency conditions. Patients with chronic pancreatitis are also at risk due to an impairment in the secretion of digestive enzymes (Trespi and Ferrieri 1999). Patients that have undergone surgery for Crohn's disease in which the ileum is damaged or removed are at increased risk of SIBO (Kholoussy al 1986). Medications that impair gastric acid secretion including PPIs are associated with an increased risk of developing SIBO (Lo and Chan 2013). More broadly, SIBO appears to be related the development of intestinal permeability, and is linked to autoimmune conditions such as fibromyalgia and roseacea (Lykova et al 2005, Goebel et al 2008, Parodi et al 2008).

The gold standard for the diagnosis of SIBO relies upon an aspirate obtained from the jejunum by endoscopy that is cultured for bacteria. The most common bacteria isolated from the small intestine of patients with SIBO are *Escherichia coli, Streptococcus, Lactobacillus, Bacteroides*, and *Enterococcus* species. A positive diagnosis of SIBO is obtained if the bacterial load is greater than 105 bacteria per millilitre, but a count as low as 103 may still suggest SIBO if the flora is predominately colonic type bacteria. The results of culturing, however, are not always representative, and false positives may occur due to contamination from the oral flora. As such, with reproducibility rates as low as 38% the reliability of this diagnostic technique has been questioned (Quigley and Quera 2006).

A non-invasive alternative to culturing jejunal aspirations are breath tests that detect the bacterial metabolism of carbohydrates to hydrogen and/or methane. This test requires that the patient fasts for a minimum of 12 hours before drinking a solution containing glucose or lactulose, and then measuring expired hydrogen and methane concentrations over a 2–3 hour period. The hydrogen breath test has been criticized, however, as it depends on the presence of hydrogen producing bacteria, and does not measure the proportion of non-hydrogen producing bacteria (Simrén M, Stotzer PO. 2006). Other similar breath tests measure the bacterial metabolism of D-xylose and glycocholic acid.

Medical treatment

SIBO is usually treated with a rotating course of antibiotics including tetracycline, amoxicillin, fluoroquinolones, metronidazole, neomycin, and rifaximin. More recently, some medical practitioners have recommended probiotics as a first line of therapy, including supplementation with *Lactobacillus casei*, *L. plantarum, L. plantarum*, *L. rhamnosus*, and *L. acidophilus*, although some probiotic species such as *L. fermentum* and *Saccharomyces boulardii* have been found to be ineffective (Quigley and Quera 2006). Another alternative to antibiotic

[42] Ghoshal UC, Shukla S, Ghoshal U. 2017. Small Intestinal Bacterial Overgrowth and Irritable Bowel Syndrome: A Bridge between Functional Organic Dichotomy. *Gut Liver.* 11(2): 196–208.

therapy is a restricted diet low in fiber to limit the fuel for bacterial fermentation, called the FODMAP diet.

Holistic treatment

SIBO relates to a fundamental weakness of digestion, typically caused by dietary and lifestyle factors such as improper food combinations, eating raw or improperly prepared food, eating too much food, eating at irregular times, and chronic mental/emotional stress. To resolve SIBO dietary modification is often suggested, and while this can be helpful, without proper treatment the diet may become limited as the patient becomes increasingly intolerant to a wide variety of foods, risking not just convenience but also nutrition and health. A good example of this is the supposed connection between SIBO and "histamine intolerance," the latter of which has long and diverse list of histamine-containing foods that must be avoided. Another frequent recommendation are probiotics and lacto-fermented foods to restore the gut microbiome, but such interventions should not be given before digestion has begun to improve, introduced to the diet in only small amounts. While dietary factors and probiotics are obviously important to restore gut health, applying just these factors alone will typically fail to resolve the condition.

A key feature of SIBO is a failure of the ileocecal sphincter to properly close and prevent the retrograde flow of bacteria into the ileum from the cecum. The ileocecal sphincter can be compromised by a number of factors, including low-grade to moderate inflammation of the cecum and appendix caused by an impairment of the colonic microbiome. A diet too low in fiber increases the risk of diverticular disease, such as cecal diverticulitis that can impair the function of the ileocecal sphincter. Likewise, a diet too high in fiber – usually commensurate with poor digestion – causes the ileocecal sphincter to remain open too often, facilitating the retrograde flow of bacteria. Apart from stretch receptors in the ileum that stimulate its opening, the tonicity of the ileocecal sphincter is regulated by the secretion of digestive hormones including gastrin (secreted in the stomach) and CCK (secreted in the duodenum). Thus, an impairment of the stomach and duodenum, as well as the biliary system that plays a role in the feedback regulation of CCK, all serve to promote a dysegulation of the ileocecal sphincter.

In Ayurveda SIBO is related to the same factors as indigestion *(ajirna)* and the accumulation of *ama*, but specifically resembles a disorder called *grahani*, a condition that often arises due to the improper treatment of gastroenteritis. The disorder relates to a relative increase in stomach function, often causing an increase in appetite, but typically a feeling of discomfort a few hours after eating due to an impairment in the alkaline secretions of the duodenum *(grahani)*. There are four basic causes of *grahani:* three related to a vitiation of each one of *doshas*, and a fourth relating to all three *doshas* in combination *(sannipata).* Mental and emotional factors such as *chinta* (worry), *bhaya* (fear), and *krodha* (anger) also play an important role in *grahani*, each of which relates to a pariclar *dosha,* i.e. worry *(kapha),* fear *(vata),* and anger *(pitta).*

In Chinese medicine, SIBO typically relates to an underlying impairment of the Spleen *qi* that is typically commensurate with Liver *qi* stagnation. In a state of health, the Liver regulates the function of the Spleen, but if the Liver *qi* stagnates and the Spleen *qi* is weak, the Liver

The Inner Alchemist
Hepatobiliary System

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Hepatic Detoxification

While routinely dismissed as a form of quackery, the term "detoxification" has a valid meaning in science, referring to an aspect of physiological function that is crucial in the maintenance of homeostasis, or an optimal physiological environment. Within medicine, the term is most often used in the context of toxicology, which is concerned with the study and removal of external toxins such as heavy metals, drugs, and alcohol. While this is perhaps the most obvious aspect of detoxification, and hence the original context in which scientists learned about it, additional research has demonstrated that it is a much broader study that contains great deal of subtly. This includes the role of diet and specific dietary components such as vitamins, minerals and phytonutrients, as well as the complex role and cumulative toxic effects of environmental toxins.

Detoxification is an inherent element of physiological function built into every cell of the body, attending not only to the biotransformation of exogenous toxins, but also to a huge diversity of internal toxins that naturally accumulate through normal metabolic processes. The body naturally produces toxins all the time, and when organs involved in detoxification, such as the liver and kidneys are impaired in their function, these wastes can accumulate to cause health issues. For example, a failure of the kidneys to secrete uric acid leads to gout, whereas if the liver cannot properly process and eliminate bilirubin, the result is jaundice. While these are obvious examples, we need to acknowledge that they do not magically appear, but exist as part of a spectrum, from nascent signs and symptoms, to a full-blown pathology. Thus despite the ease with which some health experts dismiss the role of detoxification in every day health, the concept of supporting the mechanisms of detoxification, through lifestyle, diet, and preventative medicine, is an important and rational consideration.

The term **biotransformation** refers to the chemical transformation of a biologically active molecule, usually found in the plasma of the blood, into a metabolite that is rendered inactive. Most of the processes of biotransformation occur in the liver, but other tissues and organs play

a role including the kidney, intestine, lungs and skin. On a cellular level, biotransformation takes place within the smooth endoplasmic reticulum, and is the principal organelle within hepatocytes and other cells that are responsible for metabolizing drugs and toxins. The liver plays a particularly important role in biotransformation due to its size, as well as the fact that it is the first organ to receive the blood from the digestive tract, called the "first pass effect," and contains a high concentration of drug/toxin-metabolizing enzyme systems compared to other organs.

Biotransformation takes place by a process of **enzymatic catalysis**, harnessing the activity of cellular enzymes to deactivate a drug, hormone, or toxin. The interaction of biotransformative enzymes with drugs, hormones and toxins is a relatively selective process, and a particular molecule may only be metabolized by one or a few sets of enzymes. Additionally, there are only so many proteins in the liver that can carry out these reactions, and thus when a specific enzyme system is completely saturated with a particular drug or toxin, it is working at its maximum rate. The rate at which metabolic enzymes are capable of metabolizing a certain concentration of a drug or toxin is a relatively constant and predictable, but only as long as the enzymes are not saturated. In most situations, liver enzymes are working somewhere in between their minimum and maximum rate.

In all, there are five primary patterns of biotransformative activity:

1. oxidation
2. reduction
3. hydrolysis
4. conjugation
5. transportation

The first three activities are often lumped together as **phase I detoxification,** whereas the fourth process (conjugation) is called **phase II detoxification,** and the fifth (transportation) representing **phase III detoxification**.

Phase I detoxification

Phase I detoxification is mostly performed by a family of more than 25 related enzymes called the **cytochrome P450 (CYP, cyt P450)** system, but also includes the flavin-containing monooxygenase system, alcohol dehydrogenase and aldehyde dehydrogenase, monoamine oxidase, peroxidases, esterases, and amidase epoxide hydrolase. Collectively, these phase I enzymatic reactions participate in oxidation, reduction, and hydrolysis, adding or removing a functional group of a toxic molecule, which in turn, allows it to undergo phase II detoxification.

Phase I reactions tend to make a compound more polar, and therefore water-soluble, and in some cases the detoxified compound is directly eliminated after phase I detoxification. Phase I reactions can also convert a pharmacologically inactive compound (called a prodrug) into a pharmacologically active one, such as the conversion of L-DOPA into dopamine, or the

conversion of codeine into morphine. Unfortunately, phase I detoxification can also turn nontoxic molecules into a poisonous ones, such as acetaminophen when is converted into the highly toxic N-acetyl-p-benzoquinone imine (NAPQI). Another example are the pyrrolizidine alkaloids (PAs) found in certain plant species such as comfrey *(Symphytum officinalis),* groundsel *(Senecio vulgaris),* and heliotrope *(Heliotropium arborescens)* that are converted into highly toxic pyrrole compounds.

The inducement of CYP specifically often involves the generation of free-radicals and reactive oxygen species, which if not properly deactivated in phase II reactions, can result in tissue damage. In order to prevent tissue damage, phase I reactions must not only be paired with phase II reactions, an adequate supply of antioxidant molecules is required to quench free radicals. Factors that induce phase I detoxification include:

- caffeine
- alcohol
- organochlorines
- organophosphates
- volatile organic compounds (VOCs)
- sulphonamides
- exhaust fumes
- drugs, e.g. carbamazepine, rifampin, phenytoin, phenobarbital, sulfonylureas
- herbs, e.g. St. John's Wort, grapefruit

Phase II detoxification

Most of the metabolites produced in phase I reactions are further transformed by phase II reactions, turning potential toxins into easily excretable and mostly harmless substances. The faster and more efficient phase II reactions are the less chance there is for the potentially toxic intermediate metabolites generated in Phase I reactions to cause tissue damage.

Whereas phase I reactions are in large part initiated by CYP enzymes, phase II reactions involve a series of distinctly different reactions in which the intermediate metabolites produced during phase I reactions are **conjugated**, involving the addition of a molecule to the intermediate metabolite. This process further increases the water-solubility and reduces the biological activity of the intermediate metabolite, allowing it to be harmlessly excreted in the bile or urine. Phase II reactions consist of:

- glucuronidation
- amino acid conjugation
- sulfation
- glutathione conjugation
- acetylation
- methylation

Cholelithiasis and cholecystitis

Cholelithiasis refers to the formation or presence of calculi, or "gallstones," in the gallbladder, and accounts for most extrahepatic biliary tract disorders. Typically developing over a period of years, gallstones are a mostly asymptomatic condition that is a general feature of aging, only causing significant discomfort and pain when the gallstones begin to impair or obstruct bile flow. About 10-15% of the adult population in North America suffer with cholelithiasis, with a much higher prevalence among women, First Nations groups, and Hispanics, and a lower prevalence among those of Asian and African descent. Globally, the prevalence of cholelithiasis is especially high in Westernized nations, with a lower prevalence in Asia, India, and Africa.

Gallstones are comprised of three primary materials, including cholesterol, bilirubin, and calcium, as well as smaller amounts of other substances such as protein. The vast majority of gallstones are greenish-colored **cholesterol stones**, while the remainder are brown to black-colored **pigment stones** that consist of calcium bilirubinate or other calcium salts. Pure cholesterol stones are somewhat rare, and in many cases the gallstones retrieved from a patient can be a mixture of various materials.

Cholesterol stones form due to the supersaturation of cholesterol within the bile, which eventually precipitates out in the gall bladder as solid cholesterol crystals. The mechanism for the formation of these stones appears to be related to a deficiency of 7-alpha-hydroxylase, a cytochrome P450 enzyme involved in the production of bile salts from cholesterol. Factors that increase the probability of developing cholesterol stones include obesity, rapid weight loss, metabolic syndrome, diabetes, female sex, aging, a Western diet, oral contraceptives, and family history.

The formation of **pigment stones** are unrelated to the risk factors for the development of cholesterol stones, caused instead by the accumulation of unconjugated bilirubin in the bile that precipitates as its calcium salts. The excess accumulation of bilirubin in the bile occurs with increased red blood cell destruction, and is thus more common in patients with chronic hemolytic conditions such as the thalassemias and sickle cell disease. Bilirubin accumulation can also occur with the β-glucuronidase activity of pathogenic bacteria such as *Escherichia coli* and *Bacteroides spp.* that hydrolyze glucuronic acid from conjugated bilirubin, allowing the bilirubin to be reabsorbed and directed back to the liver.

The signs and symptoms of cholelithiasis can vary, with some patients rarely complaining of any problem, whereas others complain of chronic discomfort that has relapsing and remitting nature. A transient obstruction of bile flow caused by a calculi lodged somewhere in the biliary tree typically results in **cholecystalgia**, an acute condition characterized by nausea and vomiting, along with epigastric or right upper quadrant pain that often radiates to the lower right scapula (Boas' sign). When the obstruction persists the increased inflammation results in acute **cholecystitis**. Perforation of the gall bladder and the release of bile into the peritoneum is a complication of acute cholecystitis, and usually occur as the result of a

secondary bacterial infection. Sometimes a gallstone is ejected into the common bile duct and may become lodged, temporarily obstructing the ampulla of Vater, causing the reflux of pancreatic enzymes and acute pancreatitis. The primary method used to diagnose cholelithiasis is ultrasonography.

A "porcelain" gallbladder refers to the calcification of the gallbladder itself, usually associated with chronic cholecystitis. It results from chronic inflammation that scars the internal surface of the gall bladder, which combined with the progressive calcification of the gall bladder wall, transforms the gallbladder into a porcelain-like vessel. Several studies have demonstrated an increased risk of malignancy associated with the development of a porcelain gallbladder.

Medical treatment

Given that the formation of gallstones is a somewhat natural process that occurs with aging, where they have been detected and are asymptomatic, a physician may chose to take a "watchful waiting" approach. Surgeons invariably recommend laparoscopic cholecystectomy, a procedure which surgical instruments and a video camera are inserted into the peritoneal cavity through multiple small incisions in the abdominal wall. Non-surgical treatment methods to treat cholesterol stones consist of oral bile salts such chenodeoxycholic acid (chenodiol) and ursodeoxycholic acid (ursodiol), but not in patients suffering from acute cholecystitis.

Holistic treatment

While the dramatically higher prevalence of gallbladder disease in Westernized nations is mostly related to diet and a sedentary lifestyle, the high rates of cholecystectomy in the West reflects a bias in modern medicine that the gallbladder is of secondary importance. In this way, the surgical removal of the gall bladder is considered to be a cure, despite the fact that surgery does nothing to address the underlying condition, what the German physician Rudolf Weiss called "biliary dyskinesia" (1988, 77). In his landmark text *Herbal Medicine,* Weiss further describes what he claims has been long recognized in European circles as post-cholecystectomy syndrome, a "...heterogenous condition," marked by a "regulatory failure of the...biliary system, pancreas, duodenum, and jejunum" (1988, 77). If this state of biliary dyskinesia continues untreated, it continues to manifest with symptoms such as chronic indigestion, fat malabsorption (including fat soluble vitamins and essential fatty acids), impaired liver detoxification (enhancing inflammation), and impaired biliary flow (inducing autoxicity and constipation). From a mechanistic perspective, the 7-alpha-hydroxylase deficiency that results in the supersaturation of the bile salts is a general indicator of impaired liver function, i.e. "hepatic torpor," caused by an improper diet, xenobiotic insult, excess liver burden, and a lack of bitter taste in the diet (which stimulates bile synthesis and excretion).

The holistic treatment of cholelithiasis aims to correct the underlying problem of bile synthesis and excretion. The primary issue of concern is the presence of very large stones, which if improperly induced to leave the gall bladder, can pass into the common bile duct and become lodged in the hepatopancreatic ampulla, causing a life-threatening acute pancreatitis. Thus gallstones should be identified and their size should be determined before recommending treatment. Weiss states that the key to treating gallstones is to observe three basic components: the usage of cholagogues, antispasmodics, and carminatives. Weiss notes

that cholagogues are never used in active inflammation, only when the condition has more or less achieved a latent state. Antispasmodics and carminatives are the mainstay of treatment in acute conditions (1988, 77).

A common recommendation to prevent and treat gallstones is the "olive oil liver flush." Although commonly viewed as a holistic treatment, this technique has its origins in 19th century heroic medicine, in which large amounts of olive oil (175-235 mL) were given with powerful cathartics such as calomel and podophyllin to induce purgation and excretion of the stones. Although no longer practiced in modern medicine the technique was revived by Western herbalist Frank Roberts, as described in his *Encyclopaedia of Digestive Disorders: Modern Practical Herbalism for All Digestive Disorders* (1969).

Nowadays, the most common technique consists of the patient fasting for a week on a non-fatty, vegetarian-type diet. In some techniques the patient drinks nothing but unpasteurized apple juice for the last 2-3 days, and on the evening of the last day, is given a few doses of Epsom salts, followed by a mixture of lemon juice and olive oil (~120-250 mL). After this time the patient is instructed to sleep, and after another few doses of Epsom salts in the morning, can expect the passage of the "gallstones."

In many cases, however, these are not gallstones but emulsified/saponified balls of olive oil. The sudden consumption of large amounts of olive oil promotes a large increase in the excretion of both stored bile and the pancreatic juices, and these alkaline secretions turn the unabsorbed olive oil into pale green balls of "soap."[1] While this technique isn't likely to cause an issue in healthy patients, apart from transient nausea and GI discomfort, in cholelithiasis cases there is a risk that calculi in the gall bladder may become dislodged and result in an acute obstruction of the biliary tree. In general, this is not a safe technique for patients in their 4-5th decade and beyond, due the mostly asymptomatic accumulation of calculi that happens with aging.

In some respect, the "olive oil liver flush" is similar to a technique described in Ayurveda called *snehapana*, in which increasingly larger doses of ghee are given to a patient over a period of three to seven days, the amount given and duration depending on the nature of the bowel, i.e. three days for a *mrdu* (soft) bowel; five days for *madhya* (medium) bowel; seven days for a *krura* (hard) bowel. Unlike the "olive oil liver flush," however, *snehapana* is given to enhance the unctuousness of the body and not to treat gall bladder disease, and is only given to patients that have good digestion. Another key difference is that *snehapana* is never given at night when digestion is naturally weak, but first thing in the morning. Although *snehapāna* is generally very safe, there are a number of complications that can arise with the improper dose given at the wrong time, as well as failing to observe the proper regimen. Signs and symptoms

[1] According to herbalist Michael Moore, "In the early 1980s, after recommending and teaching Robert's protocol, a PhD physiologist STRONGLY suggested that these "stones" were probably artifacts of the therapy. The next time someone passed some, I took them in a cooler to a local Santa Fe medical lab I had a working relationship with. They showed only traces of chenic and cholic bile salts, and had no discernable cholesterol content. Their educated guess was that they were saponified fatty acids...probably linoleic or oleic acid salts. They were DEFINITELY not "gallstones". I have not recommended this grim regimen since."
https://www.henriettes-herb.com/faqs/medi-3-11-gallbladder-flush.html

that indicate improper administration include a feeling of heaviness, increased mucus discharge, nausea, dyspepsia, and drowsiness.

In Ayurveda, gallstones are called *pittashmari,* and as the name denotes, are mostly caused by *pitta*, and a failure of the liver and gall bladder to properly eliminate bile *(pitta)*. In this regard, *pittashmari* is a subset of *pittavardhana,* or a general increase in *pitta*. The two other *dosha(s),* however, also play a role in gallstone formation. At its root, the underlying issue of *pittashmari* is *agnimandya,* and under the influence of *kapha*, liver function and bile flow becomes congested, and gradually the bile turns a thick, sticky sludge that accumulates in the gall bladder. As the congestion persists and bile flow becomes stagnant, *pitta* increases and causes damage to the liver. The resultant tissue damage increases *vata* and the functional capacity of the liver, essentially "drying up" its secretions, causing the gall bladder sludge to eventually coalesce into dry, hard stones. From a treatment perspective, many of the remedies used in *pittashmari* are described under another digestion disorder called *parinamashula*, which refers to digestive pain and colic *(shula)* experienced after eating *(parinama)* – a typical sign found in most patients with cholelithiasis.

In Chinese medicine, the function of the Gall Bladder is to dredge out the wastes generated by the Liver for excretion by the Large Intestine. This "dredging" function, however, becomes impaired when the Liver *qi* becomes disturbed and stagnant. This can be caused by emotional and mental states such as depression, or from chronic digestive issues, and in particular, the accumulation of Damp-Heat.

What follows is a holistic protocol to address cholelithiasis.

Dietary modifications

- a high intake of refined carbohydrates, fructose, low fiber, high fat, fast food and low vitamin C intake all increase the risk of gallstone formation
    - conversely, a high intake of monounsaturated fats, dietary fiber, olive oil, wild fish, cold-pressed PUFA, legumes, fruit, coffee, moderate alcohol consumption, and vitamin C all exert a protective role[2]
    - dietary fiber reduces the bile acid concentration in the bile by inhibiting bacterial activity that causes secondary and tertiary bile salts to be reabsorbed
    - ensure sufficient fat in diet, to stimulate bile secretion
        - avoid saturated fat and fatty meats in favor of lean meats, e.g. venison, goat, wild fish
- food combinations
    - refined starchy foods with fatty foods
        - fat delays gastric emptying, promotes gastric reflux and fermentation
        - sticky/gluey carbohydrates mixed with fat impairs action of bile
- water consumption should be increased to thin the bile (Pizzorno and Murray 1999, 1244)
- leafy greens and bitter-tasting foods (e.g. karela, bitter gourd) encourage bile secretion

[2] Di Ciaula A. et al. 2017. The Role Of Diet In The Pathogenesis Of Cholesterol Gallstones. *Curr Med Chem*. May 29

Among the conjugation pathways **glutathione conjugation** is the primary method by which intermediate metabolites are dealt with. An increased exposure to toxins as well as a poor dietary supply of glutathione cofactors promotes glutathione depletion and in increased damage from highly reactive intermediates. Glucuronidation pathways can also be reversed, for example, by the enzyme beta-glucuronidase produced by pathological gut bacteria causes conjugated toxins and hormones to become deconjugated and thus reabsorbed. The amino acids glycine, cysteine, glutamine, methionine, taurine, glutamic acid and aspartic acid are all key players in phase II reactions, and dietary deficiencies of these can cause disruptions in phase II reactions. The amino acid glutamine specifically plays a chief role in ammonia detoxification, and also helps to maintain the mucosal integrity of the gastrointestinal tract. Another important nutrient includes vitamin B6 (pyridoxal-5-phosphate, P5P), which plays a role in glutathione conjugation.

Mechanism	Enzymes	Co-factor	Location
glucuronidation	• UDP-glucuronosyltransferases	UDP-glucuronic acid	liver, kidney, intestine, lung, skin, prostate, brain
glycine conjugation	• choline acetyltransferases	glycine	liver, kidney
sulfation	• sulfotransferases	3'-phosphoadenosine-5'-phosphosulfate	liver, kidney, intestine
glutathione conjugation	• glutathione S-transferases	glutathione	liver, kidney
acetylation	• N-acetyltransferases • bile acid-CoA:amino acid N-acyltransferases	acetyl coenzyme A	liver, lung, spleen, gastric mucosa, RBCs, lymphocytes
methylation	• methyltransferase	S-adenosyl-L-methionine	liver, kidney, lung, CNS

Nuclear factor erythroid-derived 2

The synthesis of many phase II enzymes is controlled by a protein called **nuclear factor erythroid-derived 2 (Nrf2)**, which resides in the cytoplasm of a hepatocyte in an inactive state. Nrf2 is triggered by metabolism of toxins produced by CYP, after which it travels to the cell nucleus of the cell to activate genes and the synthesis of phase II enzymes. Nrf2 specifically regulates the activity of genes involved in the synthesis, activation and recycling of detoxification molecules such as glutathione, superoxide dismutase (SOD), coenzyme Q10 (CoQ10), and plays a key role in initiating heavy metal detoxification.

Phase I and II balance

In a state of health there is a natural balance between phase I and phase II reactions. If phase I reactions are more active than phase II reactions, the accumulation of reactive intermediate metabolites can lead to tissue damage and disease. Patients that suffer from this imbalance are often referred to as “pathological detoxifiers,” and are highly sensitive to fumes found in paints, perfumes and colognes, often reacting adversely to various pharmaceutical drugs, and may have a strong reaction to drinking caffeinated beverages. Sometimes a patient undergoing rapid weight loss may release stored toxins, which can create an imbalance between phase I and II reactions, leading to symptoms that are similar to a pathological detoxifier.

Phase I and II assessment

While the relative balance of phase I and phase II reactions can often be inferred by the case history, their activities can also be assessed by laboratory methods. Phase I can be assessed by the clearance of caffeine:

- low caffeine clearance
  - may indicate an impaired phase I reactions
  - increased susceptibility to toxins
- high caffeine clearance
  - may indicate overactive phase I reactions
    - excessive production of intermediate metabolites
    - glutathione depletion.

Phase II reactions can be assessed by measuring the clearance of the different metabolites generated from acetaminophen and acetyl salicylic acid (ASA). A low clearance of each of these metabolites may indicate a specific deficiency of the different enzyme systems. The following table describes these metabolites, as well as factors that may underlie deficiencies of each enzyme system, and the possible risks if the issue isn't corrected in a timely manner:

Drug	Enzyme System	Metabolite	Factors That Inhibit	Increased Risk Of
acetaminophen	conjugation	acetaminophen mercaptuate	glutathione, vitamins B_2 and B_6, selenium and zinc, or an excessive exposure to toxins, or from fasting	impaired detoxification of acetaminophen, nicotine, organophosphates and epoxides
	sulfation	acetaminophen sulfate	deficiency of cysteine, methionine, molybdenum or sulfur compounds (e.g. eggs, garlic), or the ingestion of certain drugs such as NSAIDs and tartrazine	neurotoxins, estrogens, aniline dyes, coumarin, methyl-dopa, Parkinson's and Alzheimer's diseases, rheumatoid arthritis, and neurological illness
	glucuronidation	acetaminophen glucuronide	deficiency of glucuronic acid, the usage of drugs such as acetyl salicylic acid, or from excessive oxidative stress	impaired ability to detoxify drugs such as acetaminophen, morphine, diazepam, and digitalis
ASA	glycine conjugation	salicyluric acid	deficiency of glycine and protein	increased susceptibility to various toxins

(Pizzorno and Murray 1999, 156).

The Breath of Life: Respiratory System

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    - 1500 g – qand safaid (sugar)
        - *bohat-us-saut* (hoarseness), *khushoonat-e-halaq* (pharyngitis), *waram-e-lauzatain* (tonsillitis), *waram-e-hanjara* (laryngitis), *nazla* (catarrh), *sual* (cough), *dard wa warm-e-halaq* (pharyngeal pain)
        - syrup, Rx: 20-40 mL
- Qurs-e-Sual
    - 750 g – zanjabeel *(Zingiber officinalis* rhizome)
    - 750 g – badiyan *(Illicium verum* fruit)
    - 750 g – kababchini *Piper cubeba* fruit)
    - 750 g – asl-us-soos *(Glycyrrhiza glabra* root)
    - 750 g – kakra singhi *(Pistacia intergerrima* gall)
    - 1.5 g – barg-e-arusa *(Justicia adhatoda* herb)
    - 44 kg – qand safaid (sugar)
    - 8 kg – samagh-e-arabi *(Acacia arabica* gum)
    - 8 kg – ararot *(Maranta arundinacea* root)
    - 400 g – kushta-e-abrak safaid (purified mica)
    - 200 g – sat-e-loban *(Boswellia serrata* resin)
        - Rx: 4 tablets bid
        - used in *bohat-us-saut* (hoarseness), *khushoonat-e-halaq* (pharyngitis), *nazla* (catarrh), *zukam* (coryza), *sual* (cough)

Formulations – Western herbal

- Composition Formula
    - powder, Rx: 2-3 g tid-qid
- Michael Moore's Tonsillitis Formula
    - 4 parts – redroot *(Ceanothus americanus* root)
    - 2 parts – myrrh *(Commiphora myrrha)*
    - 2 parts – bayberry *(Myrica cerifera* bark)
        - make from the individual tinctures, add 5% glycerin
        - Rx: 5 mL in 30 mL hot water every two hours as gargle

Bronchitis

Bronchitis refers to the inflammation of the large and medium bronchi. It is characterized by mucosal inflammation and the production of abundant sputum that is often mucopurulent. As the sputum accumulates in the bronchi it initiates the cough reflex, which along with the ciliated epithelia, allows the sputum to be cleared from the air passages. In some cases dyspnea results from edema and spasm of the bronchial walls. Upon auscultation the breath sounds may exhibit occasional crackling, scattered ronchi, and wheezing after coughing.

Acute bronchitis is typically the result of an acute viral URI of the pharynx, throat, and bronchial tree, sometimes with secondary bacterial infection. Fever, lymphadenopathy, myalgia and other symptoms of an upper respiratory tract infection are typically present (see **Self Defense: Nonspecific Resistance and Immunity**). Viruses that cause acute bronchitis include

adenovirus, coronavirus, influenza A and B viruses, parainfluenza virus, respiratory syncytial virus, coxsackievirus, rhinovirus, and the viruses that cause rubella and measles. Bacterial causes include *Mycoplasma pneumoniae*, *Bordetella pertussis,* and *Chlamydia pneumoniae*. Acute bronchitis may also be caused by acute exposure to various dusts, fumes and smoke. It is usually a self-limiting condition in most patients, with maximal symptoms occurring within three to five days after the onset of coughing, resolving over a two-week period. Complications usually only occur in patients with an underlying respiratory illness, including bronchiolitis and bronchopneumonia.

Chronic bronchitis is classified as a **chronic pulmonary obstructive disorder (CPOD)** that is defined as a chronic, productive cough experienced for more than a two-year period. The primary pathological features of chronic bronchitis are characterized by an increase in goblet and mucus cells with a commensurate loss of serous glands and ciliated epithlium, resulting in a thick, viscous sputum that is difficult to expectorate. With repeated inflammation there is fibrosis and a thickening of the bronchial wall, which further impairs airflow. In progressed conditions hypertrophy of the right heart ventricle (**cor pulmonae**) can occur. In most cases the patient is a smoker, although environmental pollution is another important factor and is probably an increasing trend, especially in highly congested urban areas. Nutrient deficiencies such as vitamin A, essential fatty acids and accessory antioxidants also facilitate the condition.

Medical treatment

The medical treatment of acute bronchitis is bed rest and hydration, along with antipyretics such as aspirin and acetaminophen. Antitussives used to inhibit or suppress the cough reflex do so by depressing the medullary cough center, and include drugs such as chlophedianol, levopropoxyphene, dextromethorphan, and codeine. Expectorants are also used to help expel the congested sputum from the respiratory tract by decreasing its viscosity, and include potassium iodide (side-effects include acne, coryza, erythema of face and chest, painful swelling of the salivary glands, and hypothyroidism with long term use), syrup of ipecac (nausea and vomiting), guaifenesin (generally well-tolerated), ammonium chloride, terpin hydrate, and even creosote. Demulcents are often used as an adjunct to antitussive preparations and include acacia, glycerin, honey, and sugar syrup. Antibiotics are the mainstay of treatment in purulent acute bronchitis, however, including tetracycline, erythromycin, amoxicillin or ampicillin, based on a cytological analysis of a sample of the sputum.

The medical treatment of chronic bronchitis is directed towards the removal of the cause, which may include vaccination if the condition is a sequela to seasonal URI, smoking cessation, and the use of environmental air filters. Patients may undergo allergy testing to determine the presence of potential allergens, and may be recommended to undergo desensitization therapy as a weekly injection. Symptomatic therapy consists of bronchodilators such as β_2-agonists (e.g. metaproterenol, albuterol, terbutaline, and pirbuterol) and anticholinergics (e.g. ipratropium). In come cases corticosteroids may be prescribed, either orally or topically, but there is little evidence of their benefit. Antibiotics are also used sometimes in acute exacerbations of chronic bronchitis, including increased cough, catarrh, dyspnea, and fever. In more severe forms of chronic bronchitis oxygen therapy may be administered.

Holistic treatment

In Ayurveda bronchitis is classified under the heading of *kasa* or "cough," which describes several etiological factors including the inhalation of dust and noxious fumes, excessive exercise, the chronic consumption of drying foods, and the suppression of natural urges (e.g. sneezing). There are five variants of *kasa*, including *vataja* (dry cough), *pittaja* (heat and inflammation), *kaphaja* (swelling and mucus), *kshataja* (caused by injury), and *kashaya* (caused by asthenia and wasting, i.e. tuberculosis, or *rajayakshma)*. Treated in its initial stages, bronchitis is not difficult to treat, but if allowed to continue unresolved or treated improperly, can be very difficult to cure.

In Chinese medicine cough and bronchitis are differentiated based on the etiological factors, broadly classified into that caused by extrinsic factors (i.e. Six Evils), and that which is caused by internal factors. Acute bronchitis is generally associated with external pathogens including Wind-Cold, Wind-Heat, and Heat-Dryness. Bronchitis caused by Wind-Cold is characterized by thin white sputum, coryza, aversion to cold, absence of sweat, a thin white coating on the tongue, and a floating pulse. Bronchitis caused by Wind-Heat is characterized by a cough with a thick yellow sputum, thirst, pharyngitis, coryza, fever, a thin yellow tongue coating, and a floating and rapid pulse. Bronchitis caused by Heat-Dryness is characterized by a dry cough with sputum that is difficult to expectorate, dry nose and throat, chest pan with coughing, a red-tipped tongue with a thin yellow coating, and a weak, rapid pulse.

Chronic bronchitis in Chinese medicine generally arises due to either a *yin* or *qi* deficiency of the Lungs, or is caused by a disorder of another organ system. A Lung *yin* deficiency tends to develop slowly, and the cough may moist or dry, and is sometimes accompanied by blood. The patient appears gaunt, and complains of generalized dryness, afternoon fever, malar flush, insomnia, night sweats, a red tongue, and a thread, rapid pulse. Chronic bronchitis caused by a Lung *qi* deficiency is associated with shortness of breath, weak voice, an aversion to cold, spontaneous sweating, poor immunity, a pale tongue, and a weak pulse. Given the role of the Spleen in transforming Phlegm, a deficiency of the Spleen can also give rise to chronic bronchitis, giving rise to a cough with thick white to grey-colored sputum, weak digestion, a preference for warm beverages, chest oppression, lethargy, loose motions, a swollen tongue with teeth marks and a greasy coating, and slippery pulse.

Other organs involved in bronchitis include the Liver and Kidneys. The Liver meridian runs through the chest, and in long-standing cases of Liver *qi* stagnation, can result in the ascending Liver Fire attacking the Lungs. A deficiency of Kidney *yin* can also play a role in bronchitis, and is usually commensurate with a Lung *yin* deficiency.

Ease cough, thin the mucus, and promote efficient expectoration

- mucolytic expectorants to decrease viscosity of mucus secretions (*kapha,* Wind-Cold, Spleen *yang* deficiency), e.g. ginger *(Zingiber officinalis* rhizome), cardamom *(Elettaria cardamomum* fruit), garlic (*Allium sativum* bulb), aniseed *(Pimpinella anisum* seed), cinnamon *(Cinnamomum zeylanica* bark), angelica (*Angelica archangelica* root), cayenne *(Capsicum annuum* fruit), prickly ash *(Zanthoxylum americanum* bark), *(Inula helenium* root), horseradish (*Armoracia sativa* root), pippali (*Piper longum* fruit), kartakashringi

(Pistacia intergerrima insect gall), cang er zhi (*Xanthium sibiricum*), zhi ban xia *(Pinellia ternata* prepared rhizome), chen pi *(Citrus reticulata* peel)
    - used with caution in symptoms of heat *(pitta)*
- stimulating expectorants, used in highly congested conditions with a thick profuse catarrh *(kapha,* Phlegm) e.g. heartsease *(Viola tricolor* herb), cowslip *(Primula vera* herb), daisy *(Bellis perrenis* herb), myrrh *(Commiphora myrrha* resin), balm of Gilead *(Populus trichocarpa* leaf bud), coltsfoot *(Tussilago farfara* leaf), gumweed *(Grindelia spp.* herb), marijuana *(Cannabis indica/sativa* flower), kantakari *(Solanum xanthocarpum* herb)
    - used with caution in weak/dry lungs *(vata)* and active inflammation *(pitta)*
- astringing expectorants, to check mucus production and reduce active inflammation *(pitta-kapha,* Wind-Heat, Phlegm-Heat), e.g. vasaka *(Justicia adhatoda* leaf), mullein *(Verbascum thapsus* leaf), goldenrod *(Solidago canadensis* herb), sage *(Salvia officinalis* herb), neem *(Azadirachta indica* leaf), bhunimba *(Andrographis paniculata* herb), duralambha *(Fragonia cretica* herb), sapistan *(Cordia latifolia* fruit), haridra (*Curcuma longa* rhizome), huang qin (*Scutellaria baicalensis* root),
    - possibly contraindicated with symptoms of dryness *(vata)*
- respiratory antispasmodics, to relieve spasmodic coughing and relax respiratory response, e.g. thyme *(Thymus vulgaris* herb), hyssop (*Hyssopus officinalis* herb), wild cherry (*Prunus virginiana* bark), vasaka (*Justicia adhatoda* leaf), mullein *(Verbascum thapsus* seed), coltsfoot *(Tussilago farfara* herb), gumweed *(Grindelia spp.* herb), pleurisy root *(Asclepius tuberosa* root), lobelia (*Lobelia inflata* herb), eastern skunk cabbage *(Symplocarpus foetidus* root), western skunk cabbage (*Lysichiton americanus* root), sundew (*Drosera rotundifolia* herb), bloodroot *(Sanguinaria canadensis* root), jimsonweed *(Datura* leaf/root), ma huang *(Ephedra sinica* herb), khella *(Ammi visnaga* leaf/seed), wild lettuce *(Lactuca virosa* root), opium poppy *(Papaver somniferum* immature capsule)
- respiratory demulcents, to soothe inflammation and dryness *(pitta-vata,* Lung *yin* deficiency), e.g. marshmallow *(Althaea officinalis* root), slippery elm *(Ulmus fulva* inner bark), Irish moss *(Chondrus crispus),* licorice *(Glycyrrhiza glabra* root), plantain *(Plantago spp.* herb), selfheal *(Prunella vulgaris* leaf), St. John's wort *(Hypericum perforatum* flower), chickweed *(Stellaria media* herb), shatavari *(Asparagus racemosa* root), mai men dong (*Ophiopogon japonicus* root), tian men dong *(Asparagus cochinchinensis* root), yin chai hu *(Stellaria dichotoma* root), sapistan *(Cordia latifolia* fruit)
    - avoid with symptoms of wet/cold *(kapha,* Wind-Cold, Phlegm-Cold)
- *nasya,* 2-3 gtt of sesame oil instilled and inhaled into each nostril, once daily in the morning
- *neti*, each nostril irrigated with 120 mL isotonic water, once daily in the morning
- bedside and workplace humidification, with essential oils, e.g. spruce, eucaplytus, rosemary, cedar, pine, etc.
    - topical applications, e.g. mustard *(Brassica nigra* seed plaster), kafoori *(Cinnamomum camphora* leaf balm), cayenne *(Capsicum annuum* fruit liniment or oil), balsam fir *(Abies balsamea* oleo-resin liniment, turpentine)

Resolve infection and reestablish a healthy microbiome

- antivirals, e.g. St. John's wort *(Hypericum perforatum* flower*),* osha *(Ligusticum porter/canbyi* root*),* biscuit root *(Lomatium dissectum/nudicaule* root*),* bhunimba *(Andrographis paniculata* herb*),* ban lan gen (*Isatis tinctoria* root), yu xing cao *(Houttuynia cordata* herb)
- antibacterials, e.g. goldenseal *(Hydrastis canadensis* rhizome/root*),* purple coneflower *(Echinacea angustifolia* root), wild indigo *(Baptisia tinctoria* root), garlic *(Allium sativum* bulb*),* myrrh *(Commiphora myrrha),* neem *(Azadirachta indica* leaf*),* bhunimba *(Andrographis paniculata* herb*),* haridra (*Curcuma longa* rhizome), huang lian (*Coptis chinense* root/rhizome), lian qiao (*Forsythia suspens* flower), jin yin hua (*Lonicera japonica* flower), ban lan gen (*Isatis tinctoria* root), huang qin (*Scutellaria baicalensis* root)
- antifungals, e.g. garlic *(Allium sativum* bulb*),* sweet annie *(Artemisia annua* herb), pau d'arco *(Tabebuia spp.* bark*),* barberry *(Berberis vulgaris* root*),* neem *(Azadirachta indica* leaf*),* huang lian (*Coptis chinense* root/rhizome), tulasi *(Ocimum sanctum* herb)
- probiotics, e.g. *Lactobacillus, Bifidobacterium*, live culture foods
- prebiotics, e.g. FODMAP-containing foods, chicory root, beet root, fructo-oligosaccharides (e.g. inulin)

Support detoxification and enhance mobilization of wastes

- lymphagogues, to decongest respiratory mucosa, and specifically resolve lymphadenopathy, e.g. purple coneflower *(Echinacea angustifolia* root*),* redroot *(Ceanothus americanus* root*),* pokeroot *(Phytolacca decandra* root*),* cedar *(Thuja occidentalis/plicata* leaf*),* cleavers *(Galium spp.* herb*),* red clover *(Trifolium pretense* flower)
- cholagogues and hepatotrophorestoratives to enhance liver detoxification with cholagogues and supportive nutrients (see **The Inner Alchemist**), e.g. barberry *(Berberis vulgaris* root*),* turmeric (*Curcuma longa* rhizome), guduchi (*Tinospora cordifolia* vine), bhumyamalaki *(Phyllanthus niruri* herb), huang qin (*Scutellaria baicalensis* root)
- diuretics, to dispel excess fluids via the kidneys, e.g. celery *(Apium graveolens* seed*),* cleavers *(Galium aparine* herb*),* nettle *(Urtica dioica* seed*),* goldenrod *(Solidago canadensis* herb*),* horsetail *(Equisetum arvense* herb)
- aperients to drain excess heat and fluid from the lungs, e.g. da huang *(Rheum palmatum* root), cascara sagrada *(Rhamnus purshianus* wood), trivrit *(Operculina turpethum* root), senna *(Cassia angustifolia* leaf)
- hydration and heat, e.g. showers, baths, steam baths, sweating under blankets
  - drink 1-2 liters of water daily

Support immune function and restore the lungs

- respiratory restoratives, to support and enhance lung function in chronic bronchitis, e.g. haritaki *(Terminalia chebula* fruit), amla *(Phyllanthus emblica* fruit), punarnava *(Boerhavia diffusa* root), ashwagandha *(Withania somnifera* root), ren shen *(Panax ginseng* root), American ginseng *(Panax quinquefolium* root), dang shen *(Codonopsis pilosula),* fu ling *(Poria cocos* fruiting body), gan cao *(Glycyrrhiza uralensis* root)
- immunomodulants in chronic or recurring conditions, e.g. reishi *(Ganoderma lucidum* fruiting body), maitake (*Grifola frondosa* fruiting body), huang qi (*Astragalus membranaceus),* amalaki *(Phyllanthus emblica* fruit*),* wu wei zi (*Schizandra chinense* fruit)

Mobility and Movement: Musculoskeletal System

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Western Herbal Medicine and Musculoskeletal Function

Herbal medicine has a wide range of application in the treatment and management of both acute and chronic musculoskeletal disorders. In contrast to the orientation of conventional medicine, which attempts to ameliorate a condition by supressing one or another biosynthetic pathway, herbal medicine remains true to the Hippocratic axiom of *vis medacatrix naturae,* or the "healing power of nature," attempting instead to harness the body's ability to restore itself to good health.

Orientation

Herbal medicine recognizes a broad range of therapeutic possibilities in the treatment of musculoskeletal disorders. A few of these measures have a similar intent to those used in conventional medicine, but most are unique to the traditional healing. Underlying this unique perspective is the traditional concept of vitality: what the Chinese call *qi*, Ayurveda calls *prana*, Hippocrates referred to as *vis medicatrix naturae*, and in Physiomedical terms is called the vital force. Rather than being a tangible, physical substance, the vital force represents the sum total of all positive and reconstructive homeostatic factors. Physiomedicalism teaches that disease arises when there is some obstruction to the vital force of the body, resulting in a dysregulation of homeostatic feedback mechanisms, and common problems such as chronic inflammation. Instead of seeking to inhibit or suppress this dysregulation, the herbalist seeks to resolve it by removing that which obstructs vital function.

Nutrition

Although rarely considered in modern medicine, dietary modification is an important strategy in the holistic treatment of musculoskeletal disorders, and basic nutrients such as adequate protein, minerals, and essential fatty acids are of vital importance. Important as well are a broad range of phytonutrients contained in vegetables and fruits that have a huge range of biological activities, from antioxidant effects (e.g. anthocyanidins in blueberries), to hormonal regulation (e.g. phytoestrogens in cruciferous vegetables), to simply supplying important nutrients (e.g. vitamin C, vitamin K, fiber, etc.).

Among the most prominent changes to our diet over the last 9000 years ago is an increasing reliance upon cereal grains and legumes. Such foods have been shown to promote **mineral deficiencies** through the chelating activity of phytic acid, as well as promote gastrointestinal damage through the activity of lectins.[6] This situation is made worse by the fact that most food is grown in increasingly nutrient depleted soils. A deficiency of macrominerals such as calcium and magnesium, as well as trace minerals such as silicon and boron, have important implications for musculoskeletal health, interfering with the vital activities such as muscle contraction and bone mineralization. The most prominent source of minerals in the traditional human diet is from animal sources, referring not only the skeletal muscle that most people nowadays eat exclusively, but also the cartilage, ligaments, tendons, marrow, and organ meats, as well as slow-cooked bone broths. Another vitally important source of trace minerals in the traditional diet were plant foods, especially leafy green such as wild nettle or sea vegetables such as kelp. To optimize mineral status such foods should be consumed on a regular basis.

For vegetarians, there is a greater challenge to acquire the same level of nutrient density in the diet, but it is possible. The traditional lacto-ovo vegetarian diet of India is perhaps the best example of a sustainable vegetarian diet. Milk and especially eggs are a vitally important source of nutrient-dense, bio-assimilable proteins and fats, and are also a good source of certain minerals. Additional calcium that has been clinically shown to benefit deficiency disorders such as osteoporosis can be obtained from dried/sterile eggshell powder, about 1 tsp daily.[7] While the Indian vegetarian diet is also high in mineral-chelators such as grains and legumes, traditional methods of food preparation including fermentation (e.g. *idli)* and double-cooking (e.g. dal fry) helps to reduce these antinutrient factors. In addition to such techniques, traditional Indian cuisine typically draws upon an abundance of herbs and spices, such as coriander, cumin, and ajwain, all of which dramatically boost the mineral content of the diet.

Milk is perhaps the most celebrated of foods in Indian culture, and for vegetarians that eschew eggs dairy serves as the primary source of protein in their diet. In marketing campaigns milk is frequently touted as a bone-builder due its high calcium content, but this is a gross over-simplification of the nutritional requirements of bone. When compared to other mineral sources such as bone broths or seaweed milk isn't necessarily the best choice, as the

[6] Freed DL. 1999. Do dietary lectins cause disease? *BMJ.* 318:1023-1024

[7] Schaafsma A1, van Doormaal JJ, Muskiet FA, et al. 2002. Positive effects of a chicken eggshell powder-enriched vitamin-mineral supplement on femoral neck bone mineral density in healthy late post-menopausal Dutch women. *Br J Nutr.* 87(3):267-75.

complex process of bone mineralization is not solely dependent on calcium. Magnesium for example, which competes with calcium for absorption and is comparatively low in cow's milk, plays a vitally important role in bone formation. Magnesium is an important co-factor in alkaline phosphatase, an enzyme involved in the formation of hydroxyapatite,[8] and participates in the conversion of vitamin D into its biologically active form.[9] Research has shown that magnesium supplementation promotes bone formation, prevents bone resorption, and increases the dynamic strength of bone.[10]

Contrary to the belief that milk is a bone-building food, the recommendation to drink milk on a daily basis to prevent osteoporosis has not withstood the rigors of epidemiology, which shows that populations that do not drink milk have lower rates of osteoporosis than those that do.[11] The latest research indicates that rather than high doses of calcium, vitamin D is a more important factor to maintain proper bone mass.[12] In temperate countries the population is a greater risk for vitamin D deficiency due to decreased sunlight hours during winter, an increased amount of time spent indoors compared to previous generations, and the decreased consumption of traditional foods naturally rich in vitamin D such as animal liver, bone marrow, and eggs. Government agencies have attempted to correct this by adding vitamin D_2 (ergocalciferol) to the milk supply, but the biological activity of vitamin D_2 is not even one-quarter that of cholecalciferol (vitamin D_3).[13]

Topical treatments

Topical treatments form an important mainstay in the treatment of musculoskeletal disorders in herbal medicine, which utilizes a broad range of remedies including liniments, salves, oils, plasters, fomentations, and baths. These different agents can be broadly categorized into two basic groups: rubifacients and analgesics.

Rubefacient therapy

At the heart of Physiomedicalism is the perspective that joint disease is a systemic disorder manifesting as a local "toxic" accumulation within the joints, serving as an obstructive force that impairs circulation to and from that region. This includes an interruption in the processes of nutrition and elimination, resulting in a loss of vital capacity and the progressive

[8] Iseri LT, French JH. 1984. Magnesium: Nature's physiologic calcium blocker. *Am Heart J* 108:188-93

[9] Rude RK et al. 1985. Low serum concentration of 1,25 dihydroxyvitamin D in human magnesium deficiency. *J Clin Endocrinol Metab* 61:933-40

[10] Toba Y et al. 2000. Dietary magnesium supplementation affects bone metabolism and dynamic strength of bone in ovariectomized rats. *J Nutr* 130(2):216-20

[11] Kaneki M, Hedges SJ, Hosoi T, Fujiwara S, Lyons A, Crean SJ, Ishida N, Nakagawa M, Takechi M, Sano Y, Mizuno Y, Hoshino S, Miyao M, Inoue S, Horiki K, Shiraki M, Ouchi Y, Orimo H. 2001. Japanese fermented soybean food as the major determinant of the large geographic difference in circulating levels of vitamin K2: possible implications for hip-fracture risk. *Nutrition.* 17(4):315-21

[12] Feskanich D, Willett WC, Colditz GA. 2003. Calcium, vitamin D, milk consumption, and hip fractures: a prospective study among postmenopausal women. *Am J Clin Nutr.* 77(2):504-11

[13] Vieth, Reinhold. 2001. Vitamin D Nutrition and its Potential Health Benefits for Bone, Cancer and Other Conditions. *Journal of Nutritional & Environmental Medicine.* 11, 275–291

encroachment of the disease. To overcome this dynamic measures are taken in Physiomedicalism to improve circulation and enhance the removal of this congestive element.

This notion, however, of breaking up a perceived congestive element in order to restore proper function is not unique to Physiomedicalism, but one that is common to every system of traditional medicine, all over the world. Cupping therapy, for example, which irritates a local area through suction to overcome pain and congestion, is traditionally used in areas as diverse as Scandinavia, the Middle East, and China. While this rather drastic measure to promote local circulation is often regarded with skepticism, there is good clinical data to suggest that it is effective, for example in the treatment of osteoarthritis.[14] While there is limited research on rubefacient therapy specifically, there are countless other studies examining the therapeutic use of massage or the application of counter-irritants such as cayenne pepper, all suggesting that the underlying principle of rubefacient therapy has a firm basis in science.

A major limitation in the treatment of any joint disorder is that the articular surfaces are avascular and cannot be effectively treated by internal methods alone. To support healing, rubefacients serve to enhance local vasodilation and the proliferation of proinflammatory mediators, promoting the removal of waste products and the stimulation of repair and regeneration. While almost any metabolic process characterized as "inflammatory" is considered a valid therapeutic target in modern medicine, the holistic perspective is to gently enhance and support the natural process of inflammation, to optimize and speed healing. Found not only in the treatment of articular disorders, this approach is also utilized in the treatment of disorders such as fever and diarrhea, where the orientation is manage the inflammatory process and account for any problems that might arise as a result (e.g. dehydration), rather than to suppress it's signs and symptoms.

Partly this difference between modern and traditional medicine lies in how the term "inflammation" is defined. Since the Roman physician Aulus Celsus articulated them 2000 years ago, four cardinal signs of inflammation have been identified in the Western medical tradition:

1. *calor* – heat
2. *rubor* – redness
3. *tumor* – swelling
4. *dolor* – pain

These four signs are clear indications that inflammation is present, and if any two are expressed inflammation can almost be assumed to be part of the clinical presentation. A fifth cardinal sign called *functio laesa*, or loss of function, is sometimes attributed to Claudius Galen but was likely added in the 17th century by Thomas Sydenham, "the English Hippocrates." For hundreds if not thousands of years these four (or five) cardinal signs have communicated a very precise understanding of how to identify the inflammatory response.

[14] Wang B, Liu X, Hu Z, Sun A, Ma Y, Chen Yingying, Zhang X, Liu M, Wang Y, Wang S, Zhang Y, Li Y, Shen W. 2016. Yang's pricking-cupping therapy for knee osteoarthritis: a multi-center randomized controlled trial [Article in Chinese]. *Zhongguo Zhen Jiu*. 36(2):113-8.

- Bone, Flesh and Cartilage
  - decoction, Rx: 200 mL bid-tid
  - can be applied topically as a fomentation or bath
- Rheumatic Drops
  - Rx: 5-10 mL, diluted in water, bid-tid

Gout

Gout is a group of heterogenous diseases characterized by a recurrent acute or chronic arthritis of the peripheral joints that entirely results from a sustained increase in serum uric acid levels. Due to its poor solubility, **monosodium urate crystals** begin to accumulate in and about the joints of the extremities, in the cartilage initially, and then in the bones and tendons. This initiates an inflammatory response that results in a granuloma-like tissue comprised of giant cells and activated mononuclear cells. As the condition progresses a white chalky deposit on the articular surfaces begins to accumulate. Sustained **hyperuricemia** can eventually lead to gouty arthritis of the central joints, or tissue damage to organs such as the kidneys.

A typical cause of hyperuricemia is the **impaired renal clearance of urate**, and thus gout is more common in patients that suffer from kidney disease. Alcoholics are also at significantly greater risk of impaired urate clearance as a secondary effect of excess lactic acid production by the liver, which blocks urate secretion by the kidney. A less common cause of hyperuricemia and gout is **increased purine synthesis**. Purines are nitrogen containing organic compounds that comprise the nitrogenous bases used to synthesize DNA and RNA. Increased purine synthesis seen in diseases marked by excess cellular proliferation, such as psoriasis, lymphoma and leukemia, can also lead to gout.

Hyperuricemia can also be caused by genetic mutations that code for certain enzymes involved in purine metabolism. The enzyme hypoxanthine-guanine phosphoribosyltransferase (HGPRT) functions to salvage purines from degraded DNA, and a mutation on the X-chromsome can lead to its deficiency. Referred to as **juvenile gout**, an HGPRT deficiency usually becomes evident by three to six months of age. The disorder can range from a relatively mild disorder (e.g. Kelley-Seegmiller syndrome) to severe (Lesch–Nyhan syndrome), leading to chronic gout, uric acid kidney stones, and cerebral palsy-like neural impairments including hypotonia, ataxia. A less common cause of juvenile gout is another X-linked mutation that leads to the hyperactivity of the enzyme phosphoribosylpyrophosphate synthetase (PRS) involved in the synthesis of nucleotides (purines).

Another potential cause of hyperuricemia is the impaired metabolism of **dietary purines**, found primarily in high-protein foods such as meat and meat products (especially in organ meats and the brain), fish, and shellfish. Dietary purines are generally low in plant-based foods, but can be found in some foods including dried peas, beans, cereals, and asparagus, as well as in yeasted foods (e.g. beer) and edible fungi. While healthy individuals typically have no problem metabolizing and clearing excess purines from the blood, patients with impaired

renal excretion including those with kidney disease well as alcoholics may experience hyperuricemia from eating a high protein diet.

The signs and symptoms of acute gouty arthritis often begin without much warning, and can be precipitated by relatively minor events such as injuring a joint (such as stubbing the toe), eating purine-rich meal, alcohol consumption, fatigue, and stress. The pain typically affects only one joint, which becomes progressively more severe and is often excruciating, resembling an acute infection. The most common clinical manifestation is **podagra**, an acute gouty arthritis of the metatarsophalangeal joint of the big toe. Gout is not limited to this particular manifestation, however, and may involve other joints such as the ankle, knee, wrist, and elbow. Polyarticular forms of gout are often accompanied by signs and symptoms such as fever, tachycardia, chills, malaise, and leukocytosis. During initial period the duration and severity of gout is usually limited, but in chronic conditions the inflammation becomes progressive and may eventually cause joint deformity. In many cases the condition is cyclical, with periods of remission and healing, interrupted by periods of exacerbation.

Although not widely reported, gout is significantly associated with metabolic syndrome, with upwards of 60% of gout patients displaying the clinical features of insulin resistance, truncal obesity, and hyperlipidemia.[56]

Medical treatment

The medical treatment of gout is based upon preventative methods, such as dietary and lifestyle modifications, analgesic medications to address acute pain and swelling, and long-term measures to treat hyperuricemia. The treatment of acute symptoms includes the use of NSAIDs, corticosteroids, opioids, and the alkaloid colchicine. NSAIDs such as indomethacin, ibuprofen, or naproxen are taken upon the first indications of pain and swelling and are continued for 24 hours after complete resolution of the acute attack, then tapered over a period of two to three days. Corticosteroids are also used to manage acute gout, and may be administered orally, intra-articularly, intravenously, or intramuscularly. Analgesics such as codeine or morphine are also used to mediate the pain of acute gout.

Colchicine is a phenylethylisoquinoline alkaloid derived from the corm (bulbo-tuber) and seed of the autumn crocus *(Colchicum autumnale)* that has long been used in Unani medicine as a treatment for gout and arthritis. Colchicine was first isolated by the French chemists P. S. Pelletier and J. B.Caventou in 1820, and has been used for many years as an unapproved drug in the US. In 2009, however, colchicine finally won FDA approval in the United States as a treatment for acute gout. Although the exact mechanism of its action has not been completely ascertained, its therapeutic action is thought to involve a reduction in lactic acid production by leukocytes, resulting in a decrease in uric acid deposition, and a reduction in phagocytosis and the inflammatory response. Colchicine also inhibits microtubule polymerization by binding to tubulin, serving as a mitotic poison that leads to apoptosis, and hence, has attracted the interest of cancer researchers. Despite its efficacy, as the purified alkaloid colchicine is poorly tolerated in up to 80% of patients, mostly complaining of gastrointestinal effects including nausea, vomiting, and diarrhea.

[56] Beyl RN, Hughes L, Morgan S. 2016. Update on Importance of Diet in Gout. *Am J Med.* 129(11):1153-1158.

The medical treatment of chronic gout involves the use of low-dose colchicine, given along with urate-lowering drugs such as probenecid and allopurinol. Probenecid acts to inhibit the tubular reabsorption of urate in the kidneys, thus increasing the urinary excretion of uric acid and decreasing serum urate levels. Allopurinol acts on purine catabolism, reducing the production of uric acid by inhibiting xanthine oxidase, the enzyme responsible for the conversion of hypoxanthine to xanthine, and xanthine to uric acid, the end product of purine metabolism. Despite their efficacy, urate-lowering drugs are potentially toxic and patients need to be closely monitored. Side effects of probenecid include possible precipitation of an acute gouty attack, renal calculi, rashes and gastrointestinal problems. Side effects from allopurinol include a rash, gastrointestinal problems, headache, urticaria and interstitial nephritis. Allopurinol in particular can promote a hypersensitivity syndrome associated with fever, bone marrow suppression, hepatic toxicity, and renal failure. Any indication of a rash while taking allopurinol is indication for its discontinuation.

Holistic treatment

Gout has long been seen a disease of excess, typified by behaviors of perhaps its most famous victim, King Henry VIII of England. Excessive meat intake alongside alcohol consumption is a primary cause of gout, and given the proclivity of men to prefer such items while engaging in typically male activities, such as eating chicken and ribs at the local pub while cheering on their favorite sports teams, it is no wonder than men suffer from gout disproportionately. The idea that gout is a disease of excess is supported by the epidemiology, which indicates that the prevalence of gout in the Western world has increased significantly over the last 100 years, and can be directly tied to increasing affluence and wealth.

In Ayurveda gout is often equated with *vatarakta*, a clinical syndrome that includes signs and symptoms of joint pain, lethargy, skin rashes, and inflammatory nodules appearing on the affected joints. *Vatarakta* is caused by a poor diet, eating incompatible foods (e.g. milk and fish), over-eating, consuming alcohol, sleeping during the day and staying awake at night, anger, and a lack of exercise. By these factors the *rakta* (blood) becomes vitiated, causing burning sensations, and when accompanied by an increase in *vata*, results in *vatarakta*. By some definitions, however, *vatarakta* also describes other medical conditions such as systemic lupus erythematosus, which can be seen as progressive or advanced form of *vatarakta*. It is further differentiated according to signs and symptoms that indicate *vata* (stiffness, coldness, tremors, lability), *pitta* (inflammation, heat, exudation and ulceration), or *kapha* (itching, coldness, heaviness, lethargy).

From a Chinese perspective gout is a manifestation of *bi* syndrome called *tong feng*, manifesting as an obstruction of *qi* and Blood that causes joint pain, inflammation, and immobility. Given the inflammatory nature of gout, it is more often associated with Heat rather than Cold. In this sense, gout can represent a worsening of the underlying causes of *bi* syndrome described under osteoarthritis, and can relate to the obstruction of Wind, Cold, and Damp within the meridians, causing the generation of Heat. According to traditional Chinese medical theory, exogenous factors too, such as excess heat and humidity, can also provoke an attack of gout.

Within the Unani system of medicine, gout can be correlated with the disease of *niqras*, an inflammatory joint disorder caused by the accumulation of morbid materials *(mawaad-e-*

faasida) within the joints, derived from one of the four humors. Etiological factors include excessive eating, alcohol consumption, a sedentary lifestyle, excessive sexual intercourse (especially after eating), and a lack of exercise. The disease is produced with a failure of the vital faculties (i.e. the liver and kidneys) to properly expel the *mawaad,* allowing it to settle in the lower extremities. The most common cause of *niqris* is bile *(safra)* mixed with phlegm *(balgham).*

What follows is a holistic approach to the treatment of both acute and chronic gout.

Ensure proper nutrition

Dietary factors that promote gout include alcohol consumption (which blocks urate excretion) and a high purine diet. Apart from eliminating alcohol, the patient should be encouraged to eat a higher proportion of alkalizing vegetable foods (e.g. leafy greens) to dissolve and eliminate urate crystals to restore serum pH. While it is important to limit purine consumption during an acute attack, for chronic gout a very low protein diet vegan-type is not recommended. If dairy is well-tolerated an Indian-style lacto-vegetarian diet is recommended, as dairy consumption appears to have a positive effect on hyperuriciemia.[57] In sensitive patients, however, dairy consumption appears to increase the risk of gout.[58]

An often over-looked dietary cause of gout is excess fructose consumption. Research has shown that the increasing prevalence of gout in developed countries has paralleled the increase in consumption of total **fructose** and in particular, **high-fructose corn syrup (HFCS).**[59] Unlike glucose, the phosphorylation of fructose consumes ATP at high rate, liberating excess adenine, resulting in an increase in circulating uric acid levels.[60] Thus similar to purines, high fructose foods should also be avoided in acute gout, and reduced overall in the diet in the treatment of chronic conditions.

Coffee has been shown to modestly reduce hyperuriciemia. Contained within coffee, caffeine is a methylxanthine that has been experimentally shown to competitively inhibit the enzyme xanthine oxidase, an enzyme that catalyzes the oxidation of xanthine to uric acid.[61] In human clinical trials, however, this effect was also noted in decaffeinated coffee, and thus this benefit appears not to be related to caffeine.[62] Furthermore, it was only with significantly higher levels of coffee consumption (≥4 cups/day) that a reduced risk (40%–60%) of gout was noted,[63]

[57] Choi HK, Liu S, Curhan G. 2005. Intake of purine-rich foods, protein, and dairy products and relationship to serum levels of uric acid: the Third National Health and Nutrition Examination Survey. *Arthritis Rheum.* 52(1):283-289.
[58] Min KB, Min JY. 2017. Increased risk for hyperuricemia in adults sensitized to cow milk allergen. Clin Rheumatol. 36(6):1407-1412.
[59] Jamnik J, Rehman S, Blanco Mejia S et al. 2016. Fructose intake and risk of gout and hyperuricemia: a systematic review and meta-analysis of prospective cohort studies. *BMJ Open.* 6(10):e013191.
[60] Kedar E, Simkin PA. 2012. A perspective on diet and gout. *Adv Chronic Kidney Dis.* 19:392–7.
[61] Larsson SC, Carlström M. 2018. Coffee consumption and gout: a Mendelian randomisation study. *Ann Rheum Dis.* pii: annrheumdis-2018-213055.
[62] Choi HK, Curhan G. 2007. Coffee, tea, and caffeine consumption and serum uric acid level: the third national health and nutrition examination survey. *Arthritis Rheum.* 57(5):816-21.
[63] Larsson SC, Carlström M. 2018. Coffee consumption and gout: a Mendelian randomisation study. *Ann Rheum Dis.* pii: annrheumdis-2018-213055.

Skin Deep:
Integumentary system

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Traditional Perspectives on Skin Disease

Ayurveda and skin disease

Ayurveda states that the skin is formed by the metabolic activity of blood just as a layer of scum forms on the surface of heated milk as it begins to cool. Thus the skin is intimately connected with the blood, and can be seen to represent a grosser, more stable phase of blood, in which changes occur to it much more slowly. According to Ayurveda, 'blood' or *rakta*, is formed by the processes of digestion, of which the first component formed is 'plasma' (*rasa*), which in turn gives rise to *rakta*. As an extension of blood the skin records upon itself the health of the blood, which in turn is reflective of digestion, and thus the skin forms a useful and easily accessible indicator of both the blood and digestive health. When digestion is weak *(mandagni)* the result is the formation of *ama*, which is then absorbed into the blood and distributed all over the body. If the *ama* overwhelms the body's capacity to process and eliminate it, these 'toxins' remain in the blood and eventually begin to manifest in the skin. Unlike the blood, which undergoes constant filtering and purification by the liver and spleen, the skin is only cleansed or purified by the activities of blood. Thus it is something of a primary tenet in Ayurveda that in order for a skin condition to improve the blood must first be purified, after which the toxins present in the skin can be received by the blood and returned to the *koshta* (digestive tract, via the liver) for elimination.

Although the skin is intimately connected to the blood, and thus *pitta* (i.e. liver and spleen), it is also the repository of the sense of touch (*sparsha*), the *tanmatra* (subtle matter) that gives rise to the element of wind (*vayu*). In turn, wind is a primary component of *vata*, and thus the skin can also be seen to have an intimate connection

with the activities of the nervous system. In this way, *vata* afflictions such as anxiety and fear can easily manifest in the skin, causing problems with dryness and eczema.

Ayurveda recognizes six or seven layers to the skin, according to Charaka and Sushruta, respectively. When *ama* manifests in the skin it promotes the local vitiation of the *doshas* (i.e. *vata, pitta* or *kapha*), and depending on which layer the toxins are present, the *doshas* will manifest in that layer, giving rise to a disorder called *kushta* (skin disease).

The term *kushta* is generally used in Ayurveda to describe all skin disease, but has also been used to specifically describe what has since been identified as leprosy, i.e. infection with *Mycobacterium leprae* or *Mycobacterium lepromatosis*. The word *kushta* is derived from two words: *kush* and *dhatu*, referring to that which manifests on the outside of the body (i.e. the *bahya rogayana*, or 'external pathway' of disease), but whose origin is derived from within the body (i.e. the *antarmarga*, the 'inner pathway' of disease). *Kushta* manifests when the *doshas* are provoked and reflect their disturbed state in blood (*rakta*), skin (*twak*), lymph (*ambu*), and muscles (*mamsa*). In the chapter on *kushta (Ci: 7),* the *Charaka samhita* describes 12 factors that indirectly or directly promote the vitiation of the *doshas* that results in skin disease:

1. incompatible foods and beverages, or eating too much heavy, greasy food;
2. suppression of natural urges, especially vomiting;
3. exposure to excess heat or exercising after eating;
4. improper use of cooling (cold), heating (hot), or light (fasting) dietary regimens;
5. applying cold water directly after experiencing the hot sun, exercise, or fear;
6. eating during indigestion, or eating before the previous meal has been digested
7. improper use of *pancha karma* treatment;
8. acidic, salty or spicy foods;
9. excessive intake of freshly harvested grains, curd, fish, salt, sour, urad *(Phaseolus mungo)*, radish, flour products, sesame seed, milk, and jaggery;
10. sexual intercourse after eating;
11. sleeping during the day;
12. impropriety and immoral activity.

As described, the etiology of *kushta* traditionally included immoral and unethical behaviours such as insulting venerable people, stealing, lying, and committing other unethical acts. Ascribing such factors to *kushta,* however, is not unique, as these also play a role in the etiology of many other diseases, as well as the disease process more generally (i.e. *pragyaparadha*, 'intellectual error'). Given the insidious onset and progression of leprosy, however, it is easy to see how this disease and *kushta* more generally might be viewed as a kind of *karmic* retribution for immoral or unethical behaviour. It is likely that in ancient Indian society, sufferers of leprosy in whom significant disfigurement occurred were to some extent ostracized by the general population. This can be seen in the story of Ghosha in the *Rig Veda*, the daughter Rishi Kakshivan, who suffered from the disease of *kushta* since childhood and was unable to

get married.[2] Although leprosy has been and still is of particular concern in developing countries such as India, it is not so much a reflection of the efficacy of traditional treatments, but rather, a lack of access to proper treatment – whether medical or Ayurveda.[3]

Apart from the causative factors mentioned above, the *Ashtanga Hrdaya* specifically states that *krimi* (infection) is another prominent cause of *kushta*, including bacteria, fungi, helminths, maggots, etc. These organisms invade the skin or infect the blood by other routes (e.g. respiratory, oral), and manifest in the skin. Nonetheless, these infectious agents are often viewed as secondary factors, in which changes to the body's ecology through improper diet and lifestyle weaken immunity and promote infection. In particular, weak digestion and the development of *ama* is viewed as a major cause of susceptibility to infection.

Ayurveda suggests that skin diseases should be addressed in their incipient stage, to halt their progression and prevent a worsening of the condition. The longer a skin condition remains without treatment, the worse its prognosis. The premonitory symptoms of *kushta* include:

1. excessive roughness or smoothness of the skin;
2. excess or absent perspiration;
3. discoloration;
4. burning sensations;
5. itching;
6. loss of sensation;
7. pricking pain;
8. swelling of the skin;
9. giddiness;
10. severe pain upon injury;
11. indolent and improperly treated ulcers;
12. impaired skin healing and reappearance of previously healed skin lesions;
13. frequent horripilations ('goosebumps');
14. blackish discoloration of the blood.

The classical texts of Ayurveda describe seven major forms of *kushta* called the *maha kushta,* each of which corresponds to a particular *dosha* anord combinations thereof, i.e. *vata, pitta, kapha, vata-pitta, kapha-pitta, vata-kapha,* and *vata-pitta-kapha (sannipataja).* The following descriptions of each form of *kushta* are an amalgam from various sources, including the *Charaka samhita*, *Ashtanga Hrudaya*, and the *Madhava Nidanam*:

[2] Ghosha is said to have prayed continuously to the Ashvini Kumaras, and pleased with her deep sincerity and devotion, taught her a spiritual teaching called *Madhu Vidya* that cured her of her ailment and allowed her to find worthy husband.

[3] Current treatment for leprosy consists of a combination antibiotic therapy, to address drug-resistant strains of *Mycobacterium leprae.* Although little research has been carried out on the activity of Indian medicinal plants in leprosy specifically, many of them have well-described antibiotic properties (e.g. *Azadirachta indica).*

1. **Kapala**: quickly-spreading skin lesions that resemble brownish-red potshards *(kapala)* that are dry *(ruksha),* rough *(parusha),* and thin *(tanu)*, accompanied by a severe pricking pain *(toda bahula)*; caused by *vata.*
2. **Udumbara**: itchy *(kandu)* and painful *(ruja)* skin lesions that resemble the dusky-red udumbara fruit *(Ficus infectoria)*, accompanied by burning sensations *(daha)* and a brownish discoloration of the hair *(loma pinjaram);* caused by *pitta.*
3. **Mandala**: pink-colored *(shweta-rakta),* slowly-developing *(sthira),* compact *(sthyana),* oozing *(snigdha),* elevated *(utsanna),* circular *(mandala)* lesions that develop as an interconnected network *(anyonyasamsaktam);* caused by *kapha.*
4. **Rushyajivha**: toughened *(karkasham)* and painful *(savedanam)* skin lesions with a red margin *(rakta paryanta)* that surrounds a region of darkened pigmentation *(antaha shyava)*, like the tongue of the antelope *(rushya jivhwa)*; caused by *vata* and *pitta.*
5. **Pundarika**: elevated *(sotsedham)*, whitish-colored *(shweta)* skin lesions with a reddish margin *(rakta paryanta)*, resembling that of a lotus petal *(pundarika dala)*, accompanied by burning sensations *(daha)*; caused by *kapha* and *pitta.*
6. **Sidhma**: whitish-coppery *(shwetam tamram)* and thin *(tanu)* skin lesions that resemble the alabu flower *(Lagenaria siceraria)*, that become flaky *(vimunchati)* when rubbed *(ghrustam);* caused by *vata* and *kapha.*
7. **Kakana**: lesions colored like the seeds of gunja *(Abrus precatorius) (kakanantika varnam),* non-suppurating *(apakam)* and severely painful *(tivra vedanam);* non-curable; caused by all three *doshas (sannipataja).*

In addition to these seven primary forms of *kushta*, Ayurveda describes eleven minor forms called the *kshudra kushta,* including:

1. **Eka**: dry *(aswedanam)* skin lesions that involve a large area of the skin *(mahavastu),* resembling fish scales *(matsya);* caused by *vata* and *kapha.*
2. **Charmakhya**: thickened *(bahalam)* skin lesions, like the skin of an elephant *(hasti);* caused by *vata* and *kapha.*
3. **Kitibha**: dark pigmented *(shyavam)* skin lesions that are rough *(khara)* and hard *(parusha)*; caused by *vata* and *kapha.*
4. **Vaipadika**: severely painful *(tivra vedanam)* skin lesions, with cracks and fissures *(sphutanam),* affecting primarily the hands and feet *(panipada);* caused by *vata* and *kapha.*
5. **Alasaka**: itchy *(kandu)*, red *(saraga),* papular *(ganda)* skin lesions*;* caused by *vata* and *kapha.*
6. **Dadru**: itchy *(kandu),* red *(raga),* papulopustular *(pidaka)*, circular *(mandala),* and elevated *(udgata)* skin lesions; caused by *kapha* and *pitta.*
7. **Charmadala**: red *(rakta),* itchy *(kandu),* pustular *(sphota)* skin lesions, with cracking of the skin *(dalati)* and severe tenderness *(samsparsha asaha);* caused by *kapha* and *pitta.*
8. **Pama**: white *(shweta),* red *(aruna),* or blackish *(shyava)* colored skin lesions, characterized by an itchy *(kandu)*, vesicular *(pidaka)* eruption; caused by *kapha* and *pitta.*

Acne rosacea

Acne rosacea, often referred to as simply **rosacea**, is a common skin disorder that manifests as a red spotty rash typically found on the face, including the forehead, chin, nose and cheeks, but can also affect other regions of the body such as the eyes, chest and back. Sometimes referred to as the "adult acne," rosacea is more common during middle age and is three times more likely to affect women. Although a specific cause yet to be elucidated, rosacea has been linked to a variety of factors including chronic infection, poor digestion, improper diet, adverse drug reactions, and lifestyle factors. Triggers that have been reported to provoke rosacea or make it worse include sun exposure, emotional stress, hot weather, wind exposure, strenuous exercise, alcohol consumption, hot baths, cold weather, spicy foods, increased humidity, certain skin-care products and cosmetic, indoor heat, and hot beverages.[27] Chronic cases often manifest as small bumps and pustules with a generalized increase in local redness, often accompanied by red eyes, as well as burning or stinging sensations. If left untreated, chronic rosacea can lead to **telangiectasia** (dilation of superficial blood vessels) as well as **rhinophyma**, in which the nose gradually becomes bulbous and red from chronic inflammation. There is also evidence that rosacea can lead to the development of certain cancers including basal cell carcinoma and thyroid cancer.[28]

Four rosacea subtypes have been identified, and one patient may exhibit more than one subtype. These subtypes include:

1. **Erythematotelangiectatic rosacea** presents as erythema and telangiectasia, with a tendency to flush easily, and symptoms of burning, stinging, or itching. Erythematotelangiectatic rosacea is generally considered the most difficult form of rosacea to treat.
2. **Papulopustular rosacea** is similar to acne vulgaris, presenting as ongoing red papules that form into pustules that can last for several days. Papulopustular rosacea is thought to be one of the forms of rosacea that most responsive to treatment.
3. **Phymatous rosacea** is associated with an enlargement of the nose, called rhinophyma, presenting as a thickening of the skin, surface nodularities, enlargement, and telangiectasia. The condition can also affect the chin (gnathophyma), forehead (metophyma), cheeks, eyelids (blepharophyma), and ears (otophyma). Phymatous rosacea is more common in men.
4. **Ocular rosacea** affects the eyes and eyelids, presenting as local redness, inflammation, and telangiectasia. The eyes may feel dry, irritated, or gritty, burning, or stinging, with an increased sensitivity to light. Ocular rosacea

[27] Goldgar C, Keahey DJ, Houchins J. 2009. Treatment Options for Acne Rosacea. *Am Fam Physician*. 80(5):461-468.
[28] Li WQ, Zhang M, Danby FW et al. 2015. Personal history of rosacea and risk of incident cancer among women in the US. *Br J Cancer*. 113(3):520-3.

affects nearly 60 percent of patients with rosacea, and may lead to issues with visual acuity and vision loss.[29,30]

While rosacea officially remains an idiopathic disorder, there is increasing evidence that it reflects a disturbance to the skin's microbiome. The skin contains an enormous diversity of microbes, with over a 1000 different species of bacteria alone. This includes the *Propionibacteria* that plays a role in acne; the *Corynebacteria* that causes diphtheria; the *Staphylococci* that are a frequent cause of skin infection; and *Lactobacillus*, which serves as a probiotic organism in the gut. Not just bacteria, however, our skin also plays host to a diversity of other organisms including fungi such as the *Candida spp.* that causes yeast infections, *Trichophyton* species that cause athlete's foot and jock itch, and the Malassezia species that cause tinea versicolor.

Understanding the complexity of the microbiome is key in the treatment of many skin diseases, and no less so in rosacea, particularly considering new research that suggests disturbances to the microbiome can result in inflammatory skin disorders. For example, *Helicobacter pylori* is a bacterial pathogen commonly implicated in peptic ulcer disease, but some research shows that this pathogen may also play a role in rosacea.[31] This is an interesting finding, because it supports an age-old assertion in traditional medicine that what is happening in the skin can be a reflection of what's happening in the gut. In a similar fashion, researchers have linked a condition called small intestine bacterial overgrowth (SIBO) to rosacea, demonstrating that when the antibiotic rifaximin is used to eliminate the bacterial pathogens from the small intestine, a significant proportion of patients with rosacea note an improvement in their condition.[32]

Apart from the issue of gut health, it has been suspected for a number of years that rosacea could be the result of an infection with a tiny parasitic mite called *Demodex* (*D. folliculorum, D. brevis*). Although a normal part of the human microbiome, when the right factors are present, the population of *Demodex* can increase, resulting in an inflammatory reaction in the skin from both bites as well as the feces produced by the mites.[33] *Demodex* in particular, thrives on the waxy sebum secreted by our skin, and when temperatures increase in the spring and summer, we produce more sebum, facilitating the growth of *Demodex*. This explains why rosacea seems to get worse with sun exposure or with an increase in temperature, but it doesn't explain why teenagers

[29] Wilkin J, Dahl M, Detmar M et al. 2004.Standard grading system for rosacea: report of the National Rosacea Society Expert Committee on the classification and staging of rosacea. *J Am Acad Dermatol.* 50(6):907-12.
[30] Goldgar C, Keahey DJ, Houchins J. 2009. Treatment Options for Acne Rosacea. *Am Fam Physician.* 80(5):461-468.
[31] Kutlubay Z, Zara T, Engin B et al. 2014. Helicobacter pylori infection and skin disorders. *Hong Kong Med J.* 20(4):317-24.
[32] Parodi A, Paolino S, Greco A, et al. 2008. Small intestinal bacterial overgrowth in rosacea: clinical effectiveness of its eradication. *Clin Gastroenterol Hepatol.* 6(7):759-64.
[33] O'Reilly N, Menezes N, Kavanagh K. 2012. Positive correlation between serum immunoreactivity to Demodex-associated Bacillus proteins and erythematotelangiectatic rosacea. *Br J Dermatol.* 167(5):1032-6.

tend not to get rosacea, why rosacea seems to increase in prevalence with age, nor why rosacea is frequently associated with dry skin.

One suggestion is that as we age, the skin becomes more dry, fragile and brittle, providing more opportunity and surface area for the *Demodex* mite to thrive.[34] Certainly this same dynamic can be observed in other skin infections, such as toenail fungus, which grows into the cracks and fissures of aging, dehydrated, and calloused skin. Likewise, it has been suggested that impaired fat metabolism as we age results in the altered production of sebum as well as other factors that encourage the growth of *Demodex*.

Another feature to consider in rosacea is the issue of altered immunity. Rather than being a distinctly separate feature of rosacea, alterations in immune function that promote the inflammation of rosacea seem to be generally linked to gut health. Although the connection may seem obtuse, over 60% of immune cells are located in the gut wall, and thus dysbiosis promotes the upregulation of inflammation by the body's immune cells. The idea that rosacea could be in part an immune disorder was given credence recently when researchers discovered that rosacea sufferers often display elevated levels of **stratum corneum tryptic enzymes (SCTE)**, in conjunction with elevated levels and the altered function of an antimicrobial peptide called **cathelicidin**. When these two components are elevated, SCTE enzymes act on cathelicidin giving rise to antimicrobial peptides that directly promote skin inflammation.

The trigger for the increased production of SCTE enzymes and cathelicidin appears to be a bacteria called *Bacillus oleronius* that has been isolated from the *Demodex* mite.[35] Antigens from *Bacillus oleronius* has been shown to alter the function of toll-like receptors (TLRs), proteins on the surface of macrophages and dendritic cells that activate non-specific mechanisms of defense against microbial pathogens. Patients with rosacea have been shown to express an elevation in TLR activity, which in turn, leads to increased cathelicidin synthesis. Interestingly, TLRs also play a role in vitamin D3 metabolism, enhancing the enzymatic conversion of the precursor $25(OH)D_3$ to $1,25(OH)_2D_3$, the active form of vitamin D_3 in the body. This is important because $1,25(OH)_2D_3$ also regulates cathelicidin production, and research has demonstrated that rosacea sufferers may have high serum vitamin D levels compared to controls,[36] providing a direct link between sun exposure and the worsening of rosacea symptoms. Although it isn't clear what factors alter TLR activity, some research suggests that the

[34] Stalder JF, Tennstedt D, Deleuran M et al. 2014. Fragility of epidermis and its consequence in dermatology. *J Eur Acad Dermatol Venereol.* 28 Suppl 4:1-18.
[35] Lacey N, Delaney S, Kavanagh K. 2007. Mite-related bacterial antigens stimulate inflammatory cells in rosacea. Br J Dermatol. 157(3):474-81.
[36] Ekiz O, Balta I, Sen BB, et al. 2014. Vitamin D status in patients with rosacea. *Cutan Ocul Toxicol.* 33(1):60-2.

glucocorticoid creams often prescribed to reduce the skin inflammation in rosacea patients enhances TLR activity, directly contributing to rosacea.[37]

Medical treatment

The most commonly prescribed topical medications for acne rosacea are metronidazole (1%) and azelaic acid (15%). As an antibiotic, metronidazole disrupts the bacterial electron transport chain, leading DNA breakage and bacterial cell death. While metronidazole's effects in human cells is not entirely known, it is thought to modulate neutrophil activity, interfering with the release of reactive oxygen species that cause tissue injury.[38] Azelaic acid is a naturally occurring dicarboxylic acid found in many plants including cannabis, cereal grains (e.g. wheat, barley, sorghum) and legumes, and is also produced *by Malassezia furfur*, a yeast that lives on normal human skin as part of the commensal flora.[39] Azelaic acid has antimicrobial properties, and is thought to exert its benefit in rosacea by inhibiting cathelicidin production.[40] Other medications used in the treatment of rosacea include tetracycline antibiotics such as doxycycline and oxytetracycline applied topically and orally, particularly for phymatous and ocular rosacea. Vascular laser therapy is used cosmetically to disintegrate superficial blood vessels in telangiectasia and rhinophyma. As a second line of treatment isotretinoin is prescribed along with other measures typically used for acne vulgaris, such as the topical application of benzoyl peroxide. For the flushing and blushing that typically accompanies rosacea alpha agonists are used topically, including brimonidine, oxymetazoline, and xylometazoline.[41]

Holistic treatment

In Ayurveda, the underlying cause of rosacea is a disorder of *rakta,* the *dhatu* of blood, and thus any factor that leads to the vitiation of *rakta* can promote rosacea. *Rakta* is most closely associated with *pitta,* and thus any *pitta*-increasing factor will tend to vitiate *rakta* and make rosacea worse, e.g. sunlight, spicy food, alcohol consumption, intense emotions, etc. *Pitta* plays a role in the flushing of rosacea, as well as its basic inflammatory nature. Impurities of the blood *(ama)* can also arise from weak digestion, caused by *kapha,* contributing to the development of the pustules observed in rosacea. *Vata* relates to factors that lead to improper digestion, such an improper diet or an unregulated lifestyle, as well as a weakening of the skin caused by aging and ongoing tissue injury, as well as emotional factors such as anxiety and fear.

In Chinese medicine, rosacea is associated with an accumulation of Heat in the Lungs, often originating from excess Heat of the Stomach caused by eating spicy food or

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[37] Shibata M, Katsuyama M, Onodera T. 2008. Glucocorticoids enhance Toll-like receptor 2 expression in human keratinocytes stimulated with Propionibacterium acnes or proinflammatory cytokines. *J Invest Dermatol.* 129(2):375-82.

[38] Jones D. 2009. Rosacea, Reactive Oxygen Species, and Azelaic Acid. *J Clin Aesthet Dermatol.* 2(1): 26–30.

[39] Ashbee HR, Evans EG. 2002. Immunology of diseases associated with Malassezia species. *Clin Microbiol Rev.* 15(1):21-57.

[40] Coda AB, Hata T, Miller J, et al. 2013. Cathelicidin, kallikrein 5, and serine protease activity is inhibited during treatment of rosacea with azelaic acid 15% gel. *J Am Acad Dermatol.* 69(4):570-7.

[41] Goldgar C, Keahey DJ, Houchins J. 2009. Treatment Options for Acne Rosacea. *Am Fam Physician.* 80(5):461-468.

consuming excess alcohol. According to Chinese medical theory, the Stomach meridian passes through the cheek area, the typical region of the face that is affected in rosacea. As the condition progresses it begins to affect Blood by giving rise to Heat Toxins that can result in the pustular form of rosacea. Given the powerful association between Blood and gynecological disorders in Chinese medicine, disturbances to menstrual function can also contribute to the pathogenesis of rosacea. If the condition remains untreated the lesions become chronic and take on a dark red color, caused by Blood stasis.

Given the multiplicity of factors involved in the development of rosacea there are a variety of factors that need to be addressed, most important of which is digestion and the gut microbiome. As mentioned previously, there is an apparent link between rosacea and dysbiotic digestive disorders such as small intestine bacterial overgrowth (SIBO), and measures to address SIBO may need to be applied concurrently (see **The Inner Fire: Digestive System**). Certain medications including antibiotics, NSAIDs, PPIs, antacids, and opioids have a negative impact on the gut microbiome and should be avoided wherever possible. Generally, a diet to reduce heat and inflammation is an important component in the treatment of rosacea, as well as ensuring that the skin is properly hydrated and nourished (e.g. mineral water, broths, infusions, EFAs). Given the autoimmune-like nature of rosacea it is also important to consider eliminating agricultural staples that provoke inflammation, as well as take measures to restore the ecology of both the gut and the skin, ensuring the eradication of the *Demodex* mite.

What follows is a review of the holistic treatment of rosacea:

Ensure proper digestion

The dietary focus in rosacea is similar to that of acne vulgaris, but with a greater focus on nourishing, anti-inflammatory foods and beverages, but not to the detriment of weakening digestion further. Very spicy foods, alcohol, and stimulants, as well as eating or drinking overly hot food and beverages should be avoided.

- avoid heat *(pitta)* increasing foods and beverages, e.g. coffee, chocolate, alcohol, nightshades (e.g. potato, tomato, eggplant), chili, garlic, ginger, and sour-tasting foods (e.g. aged yogurt and dairy foods, vinegar, citrus)
- avoid potentially antigenic foods from the diet including wheat (i.e. gluten) and dairy; consider the Paleolithic diet
- emphasize a lower carbohydrate diet, avoiding refined carbohydrates including sucrose and high fructose foods (excess fruit)
- ensure diet is rich in nourishing fats, with plenty of leafy greens and bitter foods to upregulate the liver, and correct the altered fatty acid synthesis that plays a role in abnormal sebum production in rosacea
- avoid fried foods, oils and foods rich in omega 6 fatty acids, and hydrogenated fats
- emphasize mild herbs to restore digestion, e.g. fennel *(Foeniculum vulgare* seed), coriander *(Coriandum sativum* fruit), dill *(Anethum graveolens* fruit), fresh ginger *(Zingiber officinalis* rhizome), shan zha *(Crataegus pinnatifida* fruit), chen pi *(Citrus reticulata* peel)

Water of Life:
Urinary System

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Chinese Medicine and Urinary Function

A proper understanding of the urinary system in Chinese medicine is crucial to its practice, as this system governs the vital processes of the body, nested and contained within the Kidneys *(shen).* According to chapter 36 of the *Huangdi Neijing Suwen*, the two Kidneys have different but overlapping functions, with the left Kidney associated more with the function of urination, and the right Kidney associated more with its vital functions. The urinary system in Chinese medicine also includes the Urinary Bladder *(pang guang)*, which conducts the produced urine outside the body for excretion.

Kidneys, *jing,* and *ming-men*

The vital processes of the Kidneys include the *jing*, described as the primary source of *yin* in the body, and the *ming-men*, or 'ministerial fire' that represents the basis of *yang*. As such, the Kidneys represent a region of the body where *yin* and *yang* are generated, and thus the processes of life itself. In Taoist philosophy, *jing* is one component of the *sanbao* (three jewels), comprised of *jing, qi,* and *shen.*[1] *Jing* is the vital essence, or that which gives rise to the physical body, whereas *qi* replenishes and activates the body through breath, and *shen* is the mind including spiritual influences.

The *jing* contained in the Kidneys is understood to arise from two primary sources: a pre-natal origin that represents our inherited vitality and family genetics; and a post-natal source that is generated from the diet, influenced by factors such as breathing and thinking. The function of

[1] Pronounced 'shien' (*shén,* 神), differentiating this term from *shèn* (腎), which refers to the Kidneys.

jing is to maintain the essential 'juiciness' of the body, supporting *yin* functions including the production of mucus, Blood, marrow, and the reproductive tissues.

The pre-natal *jing* accounts for factors such as our innate strength and constitution, and is an inherited synthesis of parental influences acquired during conception. The prenatal *jing* is limited in quantity and can never be replenished, and its gradual decline is associated with aging and death. In the Taoist alchemical tradition, it is the preservation of *jing* that enhances longevity, and thus measures to preserve *jing* are thus undertaken as a regular practice, such as *qi gong,* meditation, and an avoidance of sex.[2]

The post-natal *jing* is derived from our diet, and is affected by factors such as breathing, thinking, and the daily regimen. It contributes to the *yin* functions of the body, but unlike the pre-natal *jing*, the post-natal *jing* is in a continual state of flux, and can both increase as well as decrease. In this regard, Chinese medicine employs a number of strategies to enhance the post-natal *jing*, including the consumption of nourishing foods such as walnuts, chives and shellfish, and medicinal plants such as he shou wu *(Polygonum multiflorum)*, dong chong xia cao *(Cordyceps chinensis),* and wu wei zi *(Schizandra chinensis).*

The Kidneys are also the site of the *ming-men*, or the Life Gate Force, where *jing* gives rise to both *yin* and *yang.* According to the *Huangdi Neijing Suwen,* the *ming-men* is located in the right Kidney, but other sources suggest that the *ming-men* lies between the Kidneys. The *ming-men* is the motive force of the body that arises at birth when the first inhalation draws the *qi* down, becoming rooted by the *jing* in the Kidneys. This becomes the *yuan qi*, or 'original *qi*', and this is continually supplemented by the *zong qi* that accumulates in the lungs from the inhalation of air mixed with the refined essence of the ingested food. The *ming-men* is responsible for the function of *yin* and *yang* in the body, and is associated with the function of the *san jiao*, or Triple Burner, that regulates the flow of secretions and circulation between the upper, middle, and lower regions of the body. The *yang* energy that arises from the *min-meng* is vitally important to fuel the Stomach and Spleen and the processes of digestion and absorption.

With such vital processes contained within the Kidneys, they are responsible for a complex array of bodily functions. As a storehouse of *jing* the Kidneys are responsible for reproductive function, which becomes easily depleted with excess sexual activity. Reproductive issues including infertility are typically associated with a depletion of Kidney function. The Kidneys are also responsible for the production of marrow, which includes the brain and spinal cord, and in turn gives rise to the bones and hair. If the Kidneys are functioning optimally the patient feels grounded, the hair is lustrous, the bones are strong, and the mind is clear. According to traditional Chinese medical beliefs the Kidneys store the *zhi* or will, which is crucial in our capacity to affect change in the world, driving us towards the fulfillment of our destiny or purpose in life.

[2] In some Taoist mystical traditions sexual activity is undertaken as a way to enhance and preserve *jing*, with a particular focus on the inhibition of male sexual emission during coitus.

The function of the urinary system

As the primary *yin* organ, the Kidneys are associated with the water element, governing the final process of separating clear from turbid fluids. This refinement process begins within the Stomach that 'rots' the ingested food, passing this refined essence to the Spleen, and the wastes along to the Small Intestine. The Small Intestine then refines this waste further, directing the refined portion back to the Spleen, the solid wastes to the Large Intestine, and the liquid wastes to the Urinary Bladder. Within the Urinary Bladder, this liquid is cooked by the Kidney *yang*, extracting the remaining *qi* contained within the fluids, and then directs the waste fluid out of the body as urine.

As the root of *yin* and *yang,* when any disease becomes chronic it will eventually affect the Kidneys. Thus Kidney deficiency syndromes are often seen in end-stage conditions, and cannot be addressed without also addressing the underlying cause. A deficiency of Kidney *yang* will directly impair the strength of the Spleen, whereas a Spleen *qi* deficiency impacts the Kidney, impairing the production of *qi* and the transformation of fluids. As the source of the inhaled *qi,* the Lungs compliment Kidney function, but are also dependent upon the Kidneys to pull the *qi* down into the body, send the *qi* back up, and to moisten the Lungs. The Liver and Kidneys have a particularly important relationship through the shared function of *yin,* in which the Liver Blood nourishes the post-natal *jing*, and the post-natal *jing* nourishes the Liver Blood.

Disorders and treatment of the urinary system

There are two primary afflictions of the Kidneys: a Kidney *yin* deficiency and a Kidney *yang* deficiency. While these are unique syndromes, because *yin* and *yang* are mutually dependent, one syndrome can lead to the other, and thus apart from the obvious differences between symptoms of hot and cold, some symptoms very similar. Thus both Kidney *yin* and *yang* deficiency manifest as soreness and weakness of the lower back and knees. Additional symptoms of a Kidney *yin* deficiency include dizziness, ringing in the ears, hearing problems, a dry mouth and throat, burning sensations, spontaneous sweating, constipation, and seminal emission. Upon assessment the radial pulse feels weak, thin and rapid in a Kidney *yin* deficiency, and the tongue appears red within a minimal coating.

Useful herbs to address a Kidney *yin* deficiency include shu di huang *(Rehmannia glutinosa),* tu si zi *(Cuscuta chinensis* fruit), han lian cao *(Eclipta prostata* herb), nu zhen zi *(Ligustrum lucidum* fruit), and shan yao *(Dioscorea opposita* tuber). One time-honored remedy for Kidney *yin* deficiency is Liu Wei Di Huang Wan (Six Ingredient Pill), comprised of:

- 240 g – shu di huang *(Rehmannia glutinosa,* root stir-fried in wine)
- 120 g – shan zu yu *(Cornus officinalis* fruit)
- 120 g – shan yao *(Dioscorea opposita* tuber)
- 90 g – fu ling *(Poria cocos* fruiting body)
- 90 g – mu dan pi *(Paeonia suffructicosa* root)
- 90 g – ze xie *(Alisma plantago-aquatica* rhizome)

- vitamin K (in hypercalciuria), 2 mg daily
- L-methylfolate (in hyperuricosuria), 5 mg daily
- sodium bicarbonate (in hyperuricosuria, monitor urine pH; avoid in struvite calculi), 1/2-1 tsp 3 times daily

Formulations – Ayurveda

- Gokshuradi guggulu vati
  - 1.344 kg – gokshura *(Tribulus terrestris* fruit) decocted in
  - 8.064 liters – water, reduced to 4.032 liters
  - 336 g – guggulu *(Commiphora wightii* resin)
  - 144 g – Trikatu churna
  - 144 g – Triphala churna
  - 48 g – musta *(Cyperus rotundus* root/rhizome)
    - pills, Rx: 2 pills bid
- Chandraprabha vati
  - pills, Rx: 2 pills bid

Formulations – Chinese medicine

- Ba Zheng San (Eight Rectification Powder)
  - clears Heat, eliminates Damp, relieves pain
  - powder, Rx: 9 g taken with warm water
  - granules, Rx: 2-4 g bid-tid
- Te Xiao Pai Shi San (modified)
  - 100 g – jin qian cao *(Desmodium styracifolium, Lysimachia christinae* herb)
  - 75 g – hai jin sha *(Lygodium japonicum* spores)
  - 75 g – da huang *(Rheum palmatum* root)
  - 50 g – ze xie *(Pyrrosia lingua* herb)
  - 50 g – bai zhi *(Angelica dahurica* root)
  - 50 g – chuan xin lian *(Andrographis paniculatus* herb)
  - 50 g – jin yin hua *(Lonicera japonica* flower)
  - 50 g – ji xue teng *(Spatholobus suberectus* stem)
    - modified from patent formula formerly available as Te Xiao Pai Shi Wan (Specific Passwan)
    - powder, Rx: 10-15 g bid-tid in acute cases; 3-5 g bid in prevention

Chronic kidney disease

Chronic kidney disease (CKD), also referred to as **chronic renal failure (CRF),** refers to a gradual loss of kidney function and a decline in the estimated glomerular filtration rate (eGFR), with resultant increases in **urea blood nitrogen (azotemia)** and **serum creatinine**. The GFR for a healthy individual ranges between 90-120 mL min/1.73 m^2, and a decrease of this rate along with clinical signs and symptoms suggests some degree of functional kidney impairment. The most common causes of CRF or **end stage renal disease** (ESRD) include:

- diabetic kidney disease
- renal artery stenosis
- glomerular disease
- cystic kidney disease
- hydronephrosis
- congenital defects of the kidney or bladder
- acute kidney injury

CKD can manifest with numerous signs and symptoms, including hypertension, muscular spasm and convulsion, peripheral neuropathy, poor appetite, nausea and vomiting, GI ulceration, malnutrition, and asthenia. Frequently the skin has a yellowish-brown cast and is pruritic, and the mouth has an ammonia-like taste because of excess urates in the saliva. In very rare cases the skin excretes excess urates in the sweat, forming a whitish crystalline excretion called a **uremic frost** (Verrelli 2005; Berkow 1992).

In 2012 a clinical guideline for the diagnosis of CKD was published by KDIGO (Kidney Disease: Improving Global Outcomes), establishing five stages of chronic kidney disease (CKD), as follows:

- Stage 1: kidney damage with normal or increased GFR (>90 mL/min/1.73 m^2)
- Stage 2: mild reduction in GFR (60-89 mL/min/1.73 m^2)
- Stage 3a: moderate reduction in GFR (45-59 mL/min/1.73 m^2)
- Stage 3b: moderate reduction in GFR (30-44 mL/min/1.73 m^2)
- Stage 4: severe reduction in GFR (15-29 mL/min/1.73 m^2)
- Stage 5: kidney failure (GFR < 15 mL/min/1.73 m^2)[4]

Frequently, the GFR rate appears to be normal in stage 1 and 2 CKD, and thus to establish a diagnosis one or more of the following criteria should be included:

- albuminuria
  - albumin excretion >30 mg/24 hr or
  - albumin:creatinine ratio >30 mg/g [>3 mg/mmol]
- urine sediment
- electrolyte abnormalities
- histologic abnormalities
- structural abnormalities detected by imaging
- history of kidney transplantation[5]

Approximately 10% of adults in the US experience some degree of CKD, and it is the ninth leading cause of death.[6,7] The US prevalence of CKD increases with age, from around 4% by

[4] Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int Suppl. 2013. 3:1-150.
[5] Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int Suppl. 2013. 3:1-150.

early middle age, increasing to almost 50% by the age of 70.[8] In the US the incidence rate of CKD among First Nations, blacks and Hispanic Americans is significantly higher than in whites, more often associated with diabetes and hypertension. In contrast, IgA nephropathy is rare in black individuals and more common among those with First Nations and Chinese Asian ancestry, linked to respiratory tract infection and liver disease (e.g. alcoholism).[9] Sex-related distribution of CKD is similar in both sexes, although the risk of hemodialysis is significantly higher in males.[10] Patients with CKD often have low circulating levels of 25(OH)D (<15 ng/mL), which is associated with an increased risk of dialysis treatment and death.[11]

The target of CKD is the kidney nephron, each of which contributes its part to maintain the glomerular filtration rate (GFR). When kidney nephrons are damaged or destroyed, the remaining healthy nephrons can undergo hypertrophy to compensate for this loss of function. In such cases, plasma levels of urea and creatinine only demonstrate measurable increases after the GFR has declined to 50%. While this compensatory action preserves kidney function, the increase in glomerular capillary pressure from hyperfiltration can damage the capillaries, leading to glomerulosclerosis (scarring of the blood capillaries).[12] Apart from underlying disease, factors that directly contribute to progressive renal injury include:

- hypertension
- nephrotoxins[13]
  - analgesics, e.g. acetaminophen, NSAIDs
  - antimicrobials, e.g. aminoglycoside, beta-lactams, rifampin, sulfonamides, vancomycin, amphotericin B
  - antidepressants, e.g. amitriptyline, fluoxetine
  - antihistamines, e.g. diphenhydramine, doxylamine
  - antiretrovirals, e.g. adefovir, cidofovir, indinavir
  - benzodiazepines
  - cardiovascular drugs, e.g. ACE inhibitors, statins
  - chemotherapeutics, e.g. cisplatin, interferon-α

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[6] United States Renal Data System. Chapter 1: CKD in the General Population. 2015 USRDS annual data report: Epidemiology of Kidney Disease in the United States. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2015

[7] Centers for Disease Control and Prevention. Deaths and Mortality. Available at http://www.cdc.gov/nchs/fastats/deaths.htm. May 3, 2017; Accessed: Feb 6, 2019

[8] United States Renal Data System. Chapter 1: CKD in the General Population. 2015 USRDS annual data report: Epidemiology of Kidney Disease in the United States. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2015

[9] Norris KC, Agodoa LY. Unraveling the racial disparities associated with kidney disease. Kidney Int. 2005 Sep. 68(3):914-24

[10] United States Renal Data System. 2017 Annual Data Report. Available at http://www.usrds.org/adr.aspx. Accessed: Feb 7 2019

[11] Kendrick J, Cheung AK, Kaufman JS, et al. 2012. Associations of plasma 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D concentrations with death and progression to maintenance dialysis in patients with advanced kidney disease. Am J Kidney Dis. 60(4):567-75.

[12] Arora, Pradeep. Medscape: Chronic Kidney Disease. Retrieved 2/28/2011 from https://emedicine.medscape.com/article/238798

[13] Naughton, Cynthia. 2008. Drug-Induced Nephrotoxicity. *Am Fam Physician.* 78(6):743-750

  - diuretics, e.g. loops, thiazides
  - illicit drugs, e.g. cocaine, heroin, methamphetamine
  - immunosuppressants, e.g. cyclosporine, methotrexate
  - environmental toxins, e.g. carbon tetrachloride, heavy metals
  - phytochemicals, e.g. aristolochic acid
  - proton-pump inhibitors
- decreased kidney perfusion (e.g. from severe dehydration)
- proteinuria
- hyperlipidemia
- hyperphosphatemia with calcium phosphate deposition
- smoking
- uncontrolled diabetes[14]

Diabetic kidney disease

Diabetic kidney disease (DKD) or **diabetic nephropathy (DN)** is the most common cause of CKD in North America. The pathogenesis is complex, but the mechanisms responsible are related to the hormonal and metabolic abnormalities observed in poorly controlled diabetes. Similar to its role in atherosclerosis, hyperglycemia causes the glycosylation of glomerular proteins, which results in vascular endothelial damage (Berkow 1992; Rubin and Farber 1990, 476).

Renal artery stenosis

Renal artery stenosis is a form of cardiovascular disease that is characterized by a progressive narrowing of the renal arteries. This narrowing is caused by atherosclerosis, affecting primarily older individuals that typically display other features of cardio-metabolic disease including obesity and diabetes. Renal artery stenosis results in an ischemic state that promotes structural damage to the kidneys and a decrease of the glomerular filtration rate, activating the renin-angiotensinogen-aldosterone in a vicious cycle to increase systemic blood pressure.

Glomerular disease

Glomerular diseases are a diverse group of diseases that affect the glomerulus, and may be primary or secondary to systemic disease. In many ways they cannot be thought of as diseases per se but as syndromes based on specific clinical and laboratory indications. The etiology is highly diverse and often mixed, and different pathological conditions can produce the same syndromes. The major categories of glomerular disease are **nephritic syndrome** and **nephrotic syndrome**.

Nephritic syndrome

Nephritic syndrome refers to inflammation of the glomerulus, characterized by clinical signs such as hematuria, hypertension, renal insufficiency, and edema. This inflammation can originate within the kidneys or be the result of infection or injury elsewhere in the body. The most common cause of nephritic syndrome is IgA nephropathy (Berger's disease), in which

[14] Arora, Pradeep. Medscape: Chronic Kidney Disease. Retrieved 2/28/2011 from https://emedicine.medscape.com/article/238798

Dwelling in the Heart: Cardiovascular System

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Etiology, Pathology and Treatment of Cardiovascular Disease

Arterial disease, atherosclerosis and hypertension

Arterial disease accounts for the vast majority of patients that suffer from cardiovascular disease, and may be accompanied by diseases of the veins and heart. The primary arterial disease is **atherosclerosis**, a progressive disease of large and medium large arteries marked by the formation of **plaques** or **atherosclerotic lesions** in the endothelium. The term **arteriosclerosis** refers to the same pathological process, but is used when discussing the atherosclerotic lesions that can occur in the smaller arterioles. The major complications of atherosclerosis include **ischemic heart disease, myocardial infarction**, and **gangrene** of the extremities. Atherosclerosis is the **leading cause of death** in North America,[17] and has been on a steady rise since the 20th century.

Pathogenesis of atherosclerosis

Atherosclerotic plaques form in the **tunica intima** of **elastic** and **muscular arteries** as a result of the proliferation of intimal smooth muscle cells and the accumulation of fat. As the lesion develops smooth muscle cells release cytokines that stimulates the accumulation of mononuclear phagocytes, lymphocytes and neutrophils in the tunica intima. As the lesion progresses the endothelium ruptures and platelets begin to adhere to it. Eventually small

[17] Roth GA, Johnson C, Abajobir A et al. 2017. Global, Regional, and National Burden of Cardiovascular Diseases for 10 Causes, 1990 to 2015. *J Am Coll Cardiol.* 70(1): 1–25.

capillaries penetrate the vessel wall and supply blood to the plaque, almost like a kind of malignant tumor (Rubin and Farber 1990, 355-369).

There are a variety of hypotheses that describe the process of atherosclerotic plaquing. While there is good post-mortem evidence of what components comprises a plaque, and now better data on the risk factors for developing atherosclerosis, the actual mechanisms of how the plaque is formed is severely limited by our inability to actually observe this process *in vivo.* As a result there are several different theories that describe the mechanism of plaquing. Some of these theories are complimentary and some are antagonistic to each other. The most commonly held belief among the medical profession is the **insudation hypothesis**, which states that the lipid found in plaques is derived from plasma lipoproteins, specifically **low density lipoproteins (LDL).** This theory states that the atherosclerotic lesion begins with a mutation of a smooth muscle cell, perhaps from chemical mutagens or microbial pathogens, resulting in focal regions of accumulation. Macrophages then scavenge LDL in the blood and transport the lipid directly into the tunica intima of the blood vessel. Additional damage to the lesion exposes circulating platelets to subendothelial collagen, which promotes the release of cytokines, chemokines, and biological response modifiers by platelets and local macrophages, stimulating the proliferation of smooth muscle cells that makes the lesion larger. With continued insudation of fat into the lesion by macrophages it eventually undergoes degeneration, and the surface of the plaque begins to ulcerate resulting in the formation of a thrombus on the injured luminal surface (Rubin and Farber 1990, 355-369).

Whatever the cause or causes, the initial lesions found in atherosclerosis are thought to be **fatty streaks**: flat or slightly elevated lesions that contain lipid. Histologically, these streaks are comprised of lipid containing macrophages referred to as **foam cells,** formed when circulating monocytes are recruited to these fatty deposits in the blood vessel walls. Monocytes then penetrate into the arterial wall due to increased endothelial permeability, and once in the subendothelial space, enlarge into macrophages. Here they initiate a variety of inflammatory processes, binding to and internalizing circulating LDL particles and storing these as liquid droplets – giving the cells a "foamy" appearance. These fatty streaks often occur at the branch points of arteries, thought to be an adaptive response to hemodynamic stress.

After the appearance of foam cells, the progression of an atherosclerotic plaque is facilitated by the secretion of cytokines, chemokines, and chemo-attractants (e.g. IL-1, IL-6, TNF), which recruit more monocytes, lymphocytes, mast cells, and neutrophils into the arterial wall. An **atheroma** develops with the local necrosis of macrophages and smooth muscle cells, inducing further inflammation and the accumulation of extracellular lipid, disrupting the normal architecture of the intima. Over time a **fibrous cap** develops over a necrotic fatty core, resulting in the formation of a **fibroatheroma**. These lesions appear as early as 15 to 30 years of age, and if the factors responsible for their formation aren't resolved, continue to develop with aging. Eventually the fibrous cap may become thin and weak, making it susceptible to rupture, resulting in the formation of a thrombus. These lesions typically appear by about the fifth or sixth decade of life, just before the peak incidences for myocardial infarction and

stroke. In many cases these ruptures heal over but may rupture again, resulting in a cycle of rupture, thrombosis, and healing.[18, 19,20]

Calcium deposition into the arterial wall is a feature throughout the development of a fibroatheroma, initially arising as small aggregates that later develop into large nodules. The mechanism of calcification is driven by the breakdown of foam cells and vascular smooth muscle cells, promoting the release of pro-inflammatory cytokines that activate calcific pathways similar to bone calcification, including the expression of major pro-osteogenic factors (e.g. osteocalcin, osteoprotegerin, osteopontin, etc.). This results in intimal cells undergoing osteogenic differentiation, secreting proteoglycans, collagen, and elastic fibers into the extracellular matrix, causing a thickening and stiffening of the artery, serving as a nidus for calcium crystal deposition. Calcification of the soft tissues results in further mechanical stress and injury, and drives the progression of the disease as a self-perpetuating cycle. The net result of all these changes is the occlusion of the blood vessel and the formation of emboli, both of which end up producing ischemia.[21,22]

Etiology of atherosclerosis

The causes of atherosclerosis are still not completely understood, with many convoluted and complicated mechanisms described. In this lesson we will examine the most commonly held belief among medical professionals, as well alternatives to this perspective.

Modern medicine has defined several risk factors for the development of atherosclerosis, some of which may or may not prove to be entirely true. All of these risk factors are based on a statistical analysis of the data called **epidemiology**, a process that helps to inform researchers of associations between certain factors and the incidence of disease. Although this process may identify groups in a population that are particularly vulnerable to a particular disease, it cannot indicate if a particular person will get a particular disease, and thus cannot take the place of an accurate, individualized health assessment. The caveat with epidemiology is that association does not prove causation.

The approach now utilized in the prevention and treatment of heart disease is in large part based upon the **Framingham Heart study**, a cohort study over 5000 adult men and women from the town of Framingham, Massachusetts. Begun in 1948, the participants of the study were analyzed for patterns related to the development of **cardiovascular disease (CVD)**. A second generation cohort study was begun in 1971, involving a similar number of participants comprised of the original participants' adult children and their spouses, and a third generation cohort study was implemented in 2002. Given the duration and number of participants

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[19] Burke AP, Kolodgie FD, Farb A et al. 2001. Healed plaque ruptures and sudden coronary death: evidence that subclinical rupture has a role in plaque progression. *Circulation.* 103: 934–940
[20] Virmani R, Kolodgie FD, Burke AP et al. 2000. Lessons from sudden coronary death: a comprehensive morphological classification scheme for atherosclerotic lesions. *Arterioscler Thromb Vasc Biol.* 20:1262–1275
[21] Faggiano P, Dasseni N, Gaibazzi N et al. 2019. Cardiac calcification as a marker of subclinical atherosclerosis and predictor of cardiovascular events: A review of the evidence. *Eur J Prev Cardiol.* 0(00):1-14
[22] Chistiakov DA, Myasoedova VA, Melnichenko AA et al. 2017. Calcifying Matrix Vesicles and Atherosclerosis. *Biomed Res Int.* 2017:7463590.

involved in the study, the Framingham Heart Study has proved to be a rich source of data for all kinds of researchers, who use a number of different methods to analyze the data and identify risk factors for CVD, including **high blood pressure**, **high blood cholesterol**, **smoking, obesity, diabetes**, and **physical inactivity**. The Framingham study has also provided additional information on the effects of factors such as **blood triglyceride** and **LDL/HDL cholesterol** levels, **age**, **gender**, and **psychosocial** issues. The Framingham data, as well as the analyses and the theories derived from it, has played an important role in the development of the modern medical curriculum. In particular, the Framingham Heart Study has been influential in establishing hypertension and elevated serum cholesterol (primarily LDL cholesterol) as the most prominent risk factors for the development of CVD.

Hypertension

Hypertension is commonly observed in atherosclerosis, caused by an increase in the pressure required by the heart to pump blood through the narrowed and occluded atherosclerotic vessels. Hypertensive patients are at increased risk of myocardial infarction and stroke. There are several causes of hypertension, such as renal artery stenosis or hyperthyroidism, and these must be ruled out for a proper diagnosis. **Essential hypertension** is a term that has been given to hypertension when the cause is unknown, or cannot be directly observed, and includes the vast majority (95%) of cases. Some theories suggest that hypertension is itself caused by hyperactivity of the renin–angiotensin system, resulting in vasoconstriction and the retention of sodium and water, increasing blood volume and raising blood pressure. Another theory is suggests that hypertension is caused by hyperactivity of the sympathetic nervous system, resulting in vasoconstriction.

While hypertension as described as a risk factor for atherosclerosis, it is more accurate to suggest that essential hypertension is a diagnostic **sign** that indicates the progressive effects of arterial damage. Unfortunately what may seem to be a fairly simple argument has been for the most part ignored by the medical profession, many of whom actively encourage hypertensive patients to use medications to lower blood pressure, even though these same medications have little impact upon morbidity and mortality, and appear to interfere with normal physiological processes (Port et al 2000).

Elevated serum cholesterol

Elevated blood cholesterol and **triglycerides** are stated as being directly correlated with the development of ischemic heart disease and atherosclerosis. The hypdophobic nature of lipids in the blood means that they must be transported with protein carriers, including chylomicrons, very low density lipoproteins (VLDL), low density lipoproteins (LDL), and high density lipoproteins (HDL). **Chylomicrons** are formed by the intestinal villi, and are comprised of globules of triglycerides, phospholipids, and cholesterol covered by a protein coating. Chylomicrons are absorbed by the lacteal of a villus, transporting fats through the lymphatic system where they enter into systemic circulation at the left subclavian vein. The triglyceride component of the chylomicron is cleaved by **lipoprotein lipase** in the blood, after which it is taken up by adipose and muscle cells and stored or used as energy. This leaves a cholesterol-rich **lipoprotein remnant** that is taken up by the liver and excreted back into the intestine as bile salts, or repacked with triglycerides into **VLDL**, where it then reenters circulation. Once again, VLDL is acted upon by lipoprotein lipase, removing triglycerides from

VLDL, forming **intermediate-density lipoproteins (IDL)** that are eventually converted into cholesterol-rich **LDL**. LDL is then taken up and processed by a variety of cells, leading to the accumulation of cholesterol within these cells. Unlike VLDL and LDL, which functions to transport cholesterol to peripheral cells, **high density lipoproteins (HDL)** functions to scavenge cholesterol and return it to the liver for excretion. Thus elevated levels of serum VLDL and LDL have been associated with a greater risk of CVD because they deposit cholesterol *into* peripheral cells, which according to the insudation hypothesis, is the primary cause of atherosclerosis. In contrast, HDL is correlated with a lower risk of CVD because it thought to *remove* cholesterol from cells (Rubin and Farber 1990, 355-369).

Despite the elegance of this hypothesis and the determination of useful serum markers for the risk of cardiovascular disease (e.g. total cholesterol, VLDL, LDL, and HDL), a complete analysis of the data suggests that there are a number of problems with the idea that cholesterol is pathogenic in CVD. When it comes to the argument that dietary cholesterol promotes hypercholesterolemia, the Framingham study clearly shows that men who ate the most cholesterol essentially have the same levels of cholesterol in their blood as those who ate the least. And while Framingham does show that the highest risk of CVD is associated with total elevated serum cholesterol, those participants with low to normal levels of serum cholesterol continue to be at significant risk. And in contrast to Framingham, other cohort studies such as the Honolulu Heart program have found that low serum cholesterol levels are actually an indicator of increased mortality (Schatz et al 2001).

In part this discrepancy between high and low cholesterol is explained by the earlier failure to differentiate between high density and low density cholesterol. Once this difference is accounted for, however, there remains a significant body of research showing that elevated LDL-C is inversely associated with CVD mortality, and that elderly people with high serum LDL-C live as long or longer than those with low LDL-C.[23] The idea that LDL-C is actually not atherogenic is further bolstered by case histories where very high levels of LDL-C are not positively correlated with coronary calcification.[24] Mainstream proponents of the LDL hypothesis counter these arguments with evidence from randomized drug trials that equate a reduction in serum LDL-C with a decrease in CVD mortality. Of particular relevance here are the HMG-CoA reductase inhibitors, or **statins,** derived from red rice yeast, which are used to interrupt the synthesis of cholesterol and thus reduce LDL/cholesterol levels in the blood. While some research does show that statins reduce the risk of cardiovascular disease, a number of trials have found that statins do not consistently lower mortality rates.[25] Statins have also been shown to have number of adverse effects, and according to the largest statin survey ever conducted, 62% of patients prescribed statins discontinued the drug due to perceived side effects, including muscle pain and weakness.[26] More recent evidence suggests

[23] Ravnskov U, Diamond DM, Hama R et al. 2016. Lack of an association or an inverse association between low-density-lipoprotein cholesterol and mortality in the elderly: a systematic review. *BMJ Open.* 12;6(6):e010401.
[24] Muacevic A, Kipp JRA, Johnson W et al. 2018. A 72-Year-Old Patient with Longstanding, Untreated Familial Hypercholesterolemia but no Coronary Artery Calcification: A Case Report Monitoring. *Cureus.* 10(4): e2452.
[25] DuBroff R, de Lorgeril M. 2015. Cholesterol confusion and statin controversy. *World J Cardiol.* 7(7):404–409.
[26] USAGE Survey. Available from: http://www.statinusage.com/Pages/key-findings-and-implications.aspx

that the benefit of statins has little to do with the benefits of lowering cholesterol, but instead, promotes stabilization of the atherosclerotic lesion.[27]

Elevated serum triglycerides

Elevated serum triglyceride has also been associated with the development of atherosclerosis, although there is a continuing controversy over its role as a causative agent. Triglycerides are a major constituent of both chylomicrons and VLDL. Peak elevation in triglyceride-rich chylomicrons is observed shortly after eating a fatty meal, whereas serum VLDL levels gradually increase during the fasting state, when the liver packages triglycerides (and cholesterol) for peripheral circulation. Eventually VLDL is converted into IDL and then the atherogenic LDL, and thus elevated serum triglyceride during the fasting state is often observed in atherosclerotic patients. Although triglycerides are not absorbed into the atherosclerotic lesion itself, it is thought that triglyceride-rich lipoprotein remnants can initiate a variety of inflammatory processes independently of LDL. Called the Zilversmit hypothesis, it is thought that when VLDL is oxidized into LDL it results in the accumulation of lipotoxins including oxidized free fatty acids and lysolecithin. These in turn induce the expression of pro-inflammatory cytokines and adhesion molecules that promote macrophage cytotoxicity and increase coagulation.[28] Although described as a distinct mechanism, and thus fingered as independent causes of CVD, the observed oxidation of VLDL and LDL are part of the same overall etiological pattern. While low-fat, high-carbohydrate diet is often recommended to lower CVD risk, this diet in particular is commonly observed to elevate plasma triglyceride (TG) concentrations[29] and increase insulin resistance.[30]

Tobacco smoke and air pollution

Beyond diet there are other factors that promote vascular injury, including tobacco smoke and air pollution. Tobacco smoke contains a host of toxic compounds, including carbon monoxide (CO), pro-oxidant gases (e.g. the reactive aldehyde acrolein), polycyclic aromatic hydrocarbons (PAHs), and heavy metals.[31] Unlike other the smoke of other plants such as cannabis, however, tobacco is an inherently toxic herb, with a limited repertoire of therapeutic use, primarily as an insecticide in the topical treatment of lice, scabies, etc. In large part this toxic effect is mediated through the presence of nicotine, an alkaloid with directly toxic effects on the nervous system, inducing a state of persistent adrenergic stimulation, impairing circulation, and altering heart function. Clinical and epidemiological research also demonstrates that both short and long-term exposure to air pollution increases the risk of cardiovascular disease. Sources of pollutants include car exhaust emissions, cigarette smoke, industrial and

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[27] Libby P, Aikawa M. 2003. Mechanisms of plaque stabilization with statins. *Am J Cardiol.* 91(4A):4B-8B.

[28] Goldberg IJ, Eckel RH, McPherson R. 2011. Triglycerides and heart disease: still a hypothesis? *Arterioscler Thromb Vasc Biol*. 31(8):1716-25.

[29] Parks EJ, Krauss RM, Christiansen MP. 1999. Effects of a low-fat, high-carbohydrate diet on VLDL-triglyceride assembly, production, and clearance. *J Clin Invest.* 104(8): 1087–1096.

[30] Jeppesen J, Schaaf P, Jones C et al. 1997. Effects of low-fat, high-carbohydrate diets on risk factors for ischemic heart disease in postmenopausal women. *Am J Clin Nutr.* 65(4):1027-33.

[31] Centers for Disease Control and Prevention (US); National Center for Chronic Disease Prevention and Health Promotion (US); Office on Smoking and Health (US). How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease: A Report of the Surgeon General. Atlanta (GA): Centers for Disease Control and Prevention (US); 2010. 6, Cardiovascular Diseases. Available from: https://www.ncbi.nlm.nih.gov/books/NBK53012/

which is a well-studied arrhythmic agent that has anti-epileptic properties. Broom has mildly inebriating, sedative properties, used in the treatment of extrasystole (palpitation), arrhythmia, and tachycardia. The dose of the tincture is 15-40 gtt bid-tid. (fresh plant 1:2, dry plant 1:5).

- Sarpagandha *(Rauwolfia serpentina* root) is an Indian herb used in Ayurveda to control *vata-pitta* conditions marked by hypertension, but may also include burning sensations, hallucination (psychosis), diarrhea, colic, and insomnia. *Rauwolfia* contains several alkaloids including reserpine which inhibit the uptake and storage of monoamine neurotransmitters in the synaptic vesicles of neurons. Reserpine actually enhancing the degradation of these neurotransmitters by monoamine oxidase (MAO), and depletes the neurotransmitter pool, with an effect that appears to have that lasts days and even weeks after last administration. By dry weight the reserpine content of *Rauwolfia* is only 0.14%, however, and is complemented by a variety of other alkaloids including ajmaline and yohimbine (1.3% total alkaloid content), as well as other constituents including flavonoids that modulate activity of reserpine. For the powdered root, the dose is 1-2 g tid-tid, and for a 1:3 tincture (50%) the starting dose is 10-15 drops, up to 2 mL bid-tid.

Also called for in CHF are diuretics to relieve pulmonary and venous congestion, such as parsley *(Petroselinum crispus* root), buchu *(Barosma betulina* herb), and pipsissewa *(Chimaphila umbellata* herb), and expectorants such as pleurisy root *(Asclepius tuberosa),* mullein *(Verbascum thapsus* herb), and elecampane *(Inula helenium* root). Useful formulas include Shilajatu rasayana (pills, Rx: 2 pills twice daily) to strengthen the heart, Mrgamadasava (10 gtt. bid-tid) to alleviate pulmonary congestion, and Vatakulantaka (pills, Rx: 2 pills twice daily) to control hypertension.

Varicose veins

A **varicose vein** is an enlarged, tortuous vessel in the venous system in filled with stagnant deoxygenated blood. Unlike the arterial system that relies upon the high pressures induced by heart contraction and the wave-like peristaltic movements of the arteries, the venous system has no independent pumping mechanism of its own, instead, relying upon a series of one way valves to direct the blood back to the heart as well as the pumping activity of muscular contraction. Deoxygenated blood is collected from tissues in venous capillaries at relative low pressures, essentially oozing out of tissues and moved along by one-way valves. The blood is then directed to superficial veins and then into the deep veins that lay next to the major arteries to direct conduct the blood to the heart. Once the blood is in the deep veins the pressure can increase dramatically through the pumping effect if muscular contraction. Unlike the arteries that are can distend with an increase in pressure, the deep veins are constructed in such as way that it prevents distension, and thus the maintenance of these pressures ensure that the blood is directed upwards to the heart. The superficial veins however are constructed differently, and can become dilated when there is too high an increase in venous pressure, either because of an obstruction in venous flow, because muscular contraction is insufficient, or because of a failure of the one-way valves. Once a one-way valve fails, it promotes an increase in pressure in the local venous network, leading to the sequential failure of the other

valves. After this process has continued for some time, the superficial veins become increasingly dilated and tortuous (Berkow 1992).

Factors that promote the obstruction of venous flow include tight clothing around the waist and especially pregnancy, which as the fetus grows, compresses the veins that drain legs, causing an increase in pressure and the manifestation of varicosities. Hormones released during pregnancy facilitate this dynamic by making vessels more pliable. Other more serious causes include a thrombosis in the deep veins, and should be ruled out. The most common cause of varicose veins is physical inactivity, especially if it is complexed with the effects of gravity. Thus people who are required to stand for long periods without moving or completely contracting the legs of the muscles are much more prone to varicosities. Researchers have also pointed out the importance of accessory nutrients such as flavonoids that play a key role in the function and repair venous tissue (e.g. aesculin, derived from horse chestnut, *Aesculus hippocastanum* seed), and thus dietary deficiencies of such nutrient can be seen to promote or exacerbate this condition. While generally not all that serious of a condition, varicose veins can be very painful, and if untreated can eventually ulcerate and become infected (Berkow 1992).

Thrombophlebitis describes the inflammation and secondary thrombosis of the small veins, as part of a local reaction to bacterial infection. **Phlebothrombosis** is the same as above but is not attributable to inflammation or infection. The term **deep vein thrombosis** is the formation of a thrombus associated with decreased cardiac output and extended bed rest, typically forming in the deep veins of the iliac and femoral veins. The primary concern in such cases is that the thrombus could embolize to the lungs, and then to the brain to cause stroke (Berkow 1992).

Holistic treatment

In Ayurveda, the veins are generally under the influence of *pitta* and hence the liver, and thus abdomino-pelvic and portal congestion is an important factor in the increase of venous pressure. Varicose veins, however, are described in Ayurveda as a *vataja* condition, relating to the degeneration of the venous tissue. Measures are thus taken to correct promote bile flow and pelvic congestion, while providing the co-factors required to ensure the integrity and repair of the blood vessels. Gentle cholagogues and pelvic decongestants are used, often rich in flavonoids, to exert a gentle astringing activity to decongest the pelvis and promote blood vessel integrity, including horse chestnut (*Aesculus hippocastanum* seed), stoneroot (*Collinsonia canadensis* root), white dead nettle (*Lamium album* herb), turmeric (*Curcuma longa* rhizome), and huang qi (*Scutellaria baicalensis* root). Other botanicals that improve blood vessel integrity include hawthorn (*Crataegus oxycanthoides* fruit), bilberry (*Vaccinium myrtilis* leaf), ginkgo (*Ginkgo biloba* leaf), amla (*Phyllanthus emblica* fruit), and gotu kola (*Centella asiatica* leaf). Circulatory stimulants are also warranted, including prickly ash (*Zanthoxylum americanum* bark), ginger (*Zingiber officinalis* rhizome), krishnajiraka (*Nigella sativa* seed), and cayenne (*Capsicum annuum* fruit). These botanicals can be formulated and taken internally as well as applied externally, twice daily. Where the tissues appear dry and weak, herbs that have a trophorestorative activity can be applied, such as the medicated oil Mahanarayana taila. In Chinese medicine the health of the vessels directly relates to the strength of the Spleen, and thus Spleen Qi restoratives are used including ginseng (*Panax ginseng* root), huang qi (*Astragalus*

membranaceus root), bai zhu (*Atractylodes macrocephala* root) and dang shen (*Codonopsis pilosula* root).

Given that varicosities can relate to thrombus formation, measures should also be taken to inhibit platelet aggregation and reduce the viscosity of the blood. Specific botanicals include (*Ginkgo biloba* leaf), garlic (*Allium sativum* bulb), and myrrh (*Commiphora molmol* resin). Useful supplements include nattokinase and lumbrokinase to prevent pulmonary emboli and stroke. Trophorestorative nutrients should also be considered, including vitamins A, B, C, E and zinc, as well as direct supplementation with flavonoids. Hot and cold hydrotherapy can also be helpful (always ending cold), and regular exercise is recommended.

Anemia

The term **anemia** refers to a decrease in the numbers of **red blood cells (RBCs)** or **hemoglobin (Hb)** content caused by a limited number of mechanisms that can function independently or occur synergistically. The term anemia is often used incorrectly as a diagnosis, but like hypertension, is really a symptom of an underlying pathology. Thus different types of anemia are defined according to the pathophysiology.

The unique concave shape of an RBC increases the surface area for gas exchange. This shape also ensures that RBCs are highly deformable, and can bend in upon themselves allowing them to squeeze through the narrow openings of capillaries into the tissues. Each RBC contains approximately 280 million molecules of hemoglobin, contained in a lipid membrane and supported by a cytoskeletal network. The rate by which RBCs develop in red bone marrow is dependent upon the status of hemoglobin, which ensures the proper oxygenation of the tissues. This process is maintained by a negative feedback mechanism that is stimulated by hypoxic conditions in the affected tissues, which in turn, promotes an increase in RBC synthesis until tissue oxygen levels are restored to normal.

RBCs develop from pluripotent hematopoietic stem cells to progenitor cells, when then form into proerythroblasts, reticulocytes and then erythrocytes (RBCs) in a process requiring a variety of growth factors and cytokines including erythropoietin. Once formed, RBC precursor cells are released into circulation as reticulocytes where they remain in circulation for about a day until they lose their nucleus. This causes the center of the cell to indent and form the distinctive concave shape of a mature RBC. Since erythrocytes have no nucleus they rely upon anaerobic and aerobic glycolytic pathways for energy, and as the cell ages, the levels of these enzymes gradually decrease. After 120 days worn and damaged RBCs are destroyed by phagocytic cells in the liver and spleen. Thus the body requires that at least 1/120 the number of RBCs are produced on a daily basis to maintain homeostasis and prevent hypoxia (Berkow 1992; Rubin and Farber 1990, 553-563).

A number of conditions can cause anemia, including:

- external blood loss: e.g. trauma, injuries, menorrhagia, and stomach ulcers
- iron deficiency: iron is an important component in the production of hemoglobin

- chronic disease: any long-term disease can lead to anemia
- kidney disease: through decreased erythropoietin secretion
- pregnancy: water gain during pregnancy is thought to dilute the RBCs (hemodilution); the fetus also robs the mother of iron during pregnancy
- poor nutrition: inadequate source of dietary iron and accessory nutrients (e.g. B complex); also common in alcoholism (Berkow 1992)

More uncommon causes of anemia include bleeding disorders, liver disease, thalassemia, infection, cancer, arthritis, enzyme deficiency, sickle cell disease, hypothyroidism, toxins, or hereditary conditions.

Signs and symptoms of anemia include:

- black and tarry stools (sticky and foul smelling)
- maroon, or visibly bloody stools
- rapid heart rate
- rapid breathing
- pale or cold skin
- jaundice
- hypotension
- heart murmur
- fatigue
- dyspnea
- chest and/or abdominal pain
- weight loss
- weakness
- vertigo and fainting, especially upon standing

Apart from external blood loss from trauma or injury, the two primary metabolic mechanisms of anemia are deficient erythropoiesis and excessive hemolysis, as follows:

Deficient erythropoiesis

Anemia is often classified according to RBC morphology, which can give an indication of the cause of the anemia, and thus terms such as microcytic anemia, normochromic-normocytic anemia, and macrocytic anemia are often used. These terms describe the different kinds of anemias that are caused by deficient erythropoiesis.

Microcytic anemia indicates an alteration in heme or globin synthesis, such as in iron deficiency, thalassemia (and related Hb-synthesis defects), and anemia of chronic diseases (e.g. infection, inflammation). **Iron-deficiency anemia** is the most common anemia, and is a chronic condition characterized by small, pale RBCs and iron depletion. The most common cause is **blood loss**, from chronic bleeding (e.g. erosive gastritis), excessive menstruation, or from a developing fetus. Other prominent causes include a dietary deficiency of iron, malabsorption from intestinal damage (e.g. inflammatory bowel disease or bowel surgery), or from the excess consumption of iron-chelating agents in diet (e.g. phytates in cereals and

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Menorrhagia

Menorrhagia refers to excessive menstrual bleeding. **Functional menorrhagia** refers to heavy bleeding during an otherwise normal menstrual cycle, whereas **secondary menorrhagia** is heavy menstrual bleeding that occurs as a result of uterine fibroids (see page 50). While it is common for women to experience heavy periods on a periodic basis, any consistent pattern of heavy bleeding requires further investigation and analysis, and in difficult cases invasive measures may be warranted, including laparoscopy, D&C (dilatation and curettage), or hysteroscopy, to determine the nature of the bleeding.

There are several factors at play in functional menorrhagia. One factor relates to an eicosanoid imbalance (e.g. elevated PGI_2) caused by imbalances in n6/n3 fatty acid consumption, the net effect of which is to inhibit clotting and blood vessel constriction. Functional menorrhagia is also associated with a relative estrogen excess and progesterone deficiency, and thus apart from adjusting fatty acid balance and the intestinal microbiome, the diagnosis of functional menorrhagia requires that hormonal causes are accounted for. One simple method to measure hormonal balance is a **sympto-thermal chart**, which can be used to assess for ovulation. Using such a chart a woman records her basal body temperature first thing in the morning, before she gets out of bed. When ovulation occurs during mid-cycle there should be a noticeable rise in body temperature, and if there isn't, it may indicate that ovulation has not occurred, suggesting a progesterone deficiency (Trickey 1998, 174-79; Berkow 1805-06).

Holistic treatment of menorrhagia

The holistic treatment of menorrhagia attempts to address the underlying factors by modifying diet and lifestyle, and using herbs to disrupt and retrain the neuro-regulatory mechanisms that cause excessive bleeding. Herbs include the use of astringents to check hemorrhage, uterine tonics to promote the tone and vitality of the uterus, and in chronic conditions, longer-term measures to address the underlying anemia. Useful uterine hemostatics include dadima *(Punica granatum* rind), beth root *(Trillium erectum* root), yarrow *(Achillea millefolium* herb), san qi *(Panax notoginseng* root), shepherd's purse *(Capsella bursa pastoris* herb), lady's mantle *(Alchemilla vulgaris* herb), raspberry leaf *(Rubus spp.* herb), and cranesbill *(Geranium maculatum* root).

Uterine tonics can be broadly separated as either more estrogenic or more progesterogenic in action. Generally speaking, progesterogenic herbs that tend to support ovulation and the luteal phase of the menstrual cycle are better choices. This includes uterine tonics such as bai shao *(Paeonia lactiflora* root), false unicorn root *(Chamaelirium luteum* root), blue cohosh *(Caulophyllum thalictroides* root), partridge berry *(Mitchella repens),* and true unicorn root *(Aletris farinosa* root). Where an estrogen deficiency appears to play a role, with symptoms such as weight-loss and anemia, more estrogenic uterine tonics such as dang gui *(Angelica sinensis* root), shatavari **(***Asparagus racemosus* root), and black cohosh *(Cimicifuga racemosa)* are indicated.

In chronic menorrhagia measures typically need to be taken to address anemia (see **Dwelling in the Heart**), including the use of blood-building herbs such as shu di huang *(Rehmannia glutinosa,* root stir-fried in wine), amla *(Phyllanthus emblica* fruit), dang gui (*Angelica sinensis* root), sang zhi *(Morus alba* fruit), dadima *(Punica granatum* pericarp), and he shou wu *(Polygonum multiflorum* root). In Western herbal medicine common "blood-building" herbs include yellowdock *(Rumex crispus* root), raspberry *(Rubus idaeus* herb), and nettle *(Urtica dioica* leaf). Such herbs can be prepared as a strong decoction (1:1), strained well, and preserved with a combination of molasses, taken in tablespoon-full doses bid-tid. Traditional formulas used in Ayurveda to treat anemia include Dadimadi churna and Shilajatu rasayana vati.

Hepatics, or 'liver' herbs are another important adjunct in the treatment of functional menorrhagia, helping to relieve the pelvic congestion that often underlies this condition, enhancing the conjugation and elimination of excess estrogen. Hepatics that specifically benefit menorrhagia include chai hu *(Bupleurum falcatum* root), Oregon grape *(Mahonia aquifolium* root), yellowdock *(Rumex crispus* root), turmeric *(Curcuma longa* rhizome), dandelion *(Taraxacum officinalis* root), and stoneroot *(Collinsonia canadensis* root). By relieving pelvic congestion hepatics are also useful in the treatment of the pain that may accompany metrorrhagia, requiring the use of antispasmodic herbs including wild yam (*Dioscorea villosa* tuber), crampbark *(Viburnum opulus* root bark), or cannabis *(Cannabis sativa* flower).

Useful nutritional supplements in functional menorrhagia include ferrous gluconate (20 mg before meals, sid-tid,), GLA (3-5 g daily), EPA/DHA (2-3 g daily), and bioflavonoids such as quercitin. Important foods to emphasize in the diet are lean animal protein (especially as soups/stews), leafy green vegetables, flavonoid-rich fruits, live culture fermented foods, and dietary fiber (to improve estrogen clearance). Animal produce should be organic and/or free-

range; dairy and fatty foods should be limited. Herbs that support digestion and have a prebiotic benefit include burdock *(Arctium lappa* root), elecampane *(Inula helenium* root), and dandelion (*Taraxacum officinale* root).

Metrorrhagia

Metrorrhagia refers to menstrual bleeding that occurs outside the regular menstrual period of a woman's normal cycle, and can range from light to heavy. Also referred to as **dysfunctional uterine bleeding (DUB),** metrorrhagia can be related to the same factors as menorrhagia, or be caused by other disorders including cancer, trauma, pelvic inflammation, and infection.

Sometimes metrorrhagia occurs as a natural event in a woman's transition into menopause, but among woman over the age of 40, chronic metrorrhagia can also be a symptom of uterine cancer. Another important cause of metrorrhagia that must be ruled out is cervical dysplasia, particularly as bleeding may indicates the progression of dysplastic changes. Any patient presenting with chronic metrorrhagia should be referred to a physician for a full workup.

Metrorrhagia can be related to physical trauma, such as in rape, or with the aggravation of cervical or endometrial polyps from sexual intercourse or with medical examination. Post-coital bleeding may also be caused by cervical ectropion, in which internal cervical glandular cells are also found on the external surface of the cervix, and bleed after contact during intercourse. Cervical ectropion may occur normally with ovulation, with oral contraceptive use, or may be a congenital abnormality, and is often confused with early-stage cervical dysplasia. Other causes of metrorrhagia include chronic pelvic inflammation (see page 51), ovarian cysts (page 56), oral contraceptive use, and excessive exercise (Trickey 1998, 192-97; Berkow 1807-08).

If the above causes can be ruled out metrorrhagia is generally related to hormonal dysregulation. This includes a relative estrogen excess, which promotes the excessive stimulation and growth of the functional endometrium. This leads to the uterus becoming engorged with blood, and when accompanied by a relative progesterone deficiency, the proper development of the endometrial lining is inhibited. This results in an impairment in the network of circulatory tissues supporting the endometrial lining, increasing blood vessel fragility and the risk of bleeding.

Holistic treatment of metrorrhagia

When caused by hormonal dysregulation, the holistic treatment of metrorrhagia is similar to that of menorrhagia, using the therapies already mentioned to astringe the tissues, tone the uterus, support ovulation, and address any underlying or associated deficiencies such as anemia. When bleeding is more or less continuous, progesterogenic herbs such as chasteberry *(Vitex agnus castus* seed) and bai shao *(Paeonia lactiflora* root) can be taken throughout the cycle, even during menses (until balance is restored), whereas hemostatic herbs would be discontinued during this time. As a symptom of increased *vata,* metrorrhagia may be accompanied by some degree of emotional distress, and thus measures to reduce and balance

vata generally are indicated, including the use of nervines such as ashwagandha *(Withania somnifera* root), St. John's wort *(Hypericum perforatum* flower), vervain *(Verbena officinalis* herb), and motherwort *(Leonorus cardiaca* herb). Useful supplements include vitamin B6 (50-100 mg sid), vitamin B complex (100 mg sid), ferrous gluconate (20 mg before meals, sid-tid,), GLA (3-5 g daily), EPA/DHA (2-3 g daily), and mixed bioflavonoids (e.g. quercetin, hesperidin). Important measures to enhance the hepatic clearance of estrogen include lipotropic factors (e.g. inositol and phosphatidyl choline) and calcium-D-glucarate (500 mg sid-bid), along with an increase in pre- and pro-biotic foods.

Amenorrhea

Amenorrhea refers to the absence of menstruation in a non-pregnant or lactating woman, and is broadly separated into either primary or secondary forms. **Primary amenorrhea** describes a condition in which menstruation has not begun by late puberty, even if other signs of physical sexual maturation are evident. **Secondary amenorrhea** refers to the cessation of menstruation for more than three menstrual cycles in a post-pubescent woman.

Primary amenorrhea is characterized by an impairment in sex steroid synthesis by the ovaries, caused by a failure of the anterior pituitary gland to synthesize and release FSH and LH (hypogonadotropic), or a failure of the ovaries to respond to elevated levels of gonadotropins (hypergonadotropic). Most forms of primary amenorrhea are congenital in origin, such as Turner sydrome, but other causes include endocrinal disturbances (e.g. Cushing's syndrome, hyperprolactinema), cancer, chronic illness, and eating disorders.

Secondary amenorrhea occurs both as an aspect of reproductive dysfunction, and with menopause, as a natural event in a woman's life. With every menstrual cycle the small bundle of ovarian follicles that secretes sex hormones undergoes atresia, and this tiny portion of the ovaries turns into scar tissue. Over her lifetime, a woman will mentruate approximately 400 times, and thus by middle age a woman's ovaries will contain a significant degree of scar tissue, and will eventually stop responding to FSH and LH. After this point, any remaining estrogen is obtained in adipose tissue through the peripheral aromatization of androgens (e.g. androstenedione) synthesized by the adrenal gland. A small percentage of woman experience premature ovarian failure undergo menopause before the age 40, and is associated with issues including autoimmune disease and exposure to toxins (e.g. chemotherapy, radiation, heavy metals, etc.)

Given the importance of adipose tissue in contributing to the estrogen pool, many non-menopausal women experiencing amenorrhea have a percentage of body fat that is well below the optimal range of 22-25%. This can occur as the result of severe illness, constitutional factors, emotional stress, eating disorders, and with intensive physical exercise. A significant decrease in body fat limits the peripheral conversion of androgen into estrogen, and can also disrupt normal feedback mechanisms, decreasing the secretion of FSH and LH. A relative increase in androgen activity caused by decreased body fat also contributes to a loss of secondary female sexual characteristics, including fertility.

Amenorrhea associated with a signficant increase in serum androgen levels occurs in disorders such as polycystic ovarian disease (PCOD), adrenal hyperplasia, adrenal adenocarcinoma, steroid use (e.g. cortisone), and obesity. The signs and symptoms of androgenization include hirsutism, alopecia, acne, and elevated blood pressure. Other more rare symptoms include the deepening of the voice, clitoral enlargement, and decreased breast size. Laboratory investigation will typically demonstrate elevated serum testosterone and DHEA, although not all cases, as some occur as a result of an increased sensitivity to androgen rather than androgen excess (Trickey 1998, 217-219; Berkow 1992, 1800).

One frequent mechanism of secondary amenorrhea is GnRH inhibition, which in turn, inhibits the secretion of the gonadotropins FSH and LH. One cause of GnRH inhibition is hyperprolactinemia, or elevated levels of prolactin in the blood. Signs and symptoms of hyperprolactinemia include galactorrhea (breast milk production), menstrual irregularities, and decreased bone density. Possible causes of hyperprolactinemia-induced amenorrhea include pituitary tumors, hypothyroidism, prolonged stress, excessive breast stimulation (Chinese "Deer" exercises), and excessive exercise. A number of drugs can also induce hyperprolactinemia including phenothiazines, dopamine antagonists (e.g. antipsychotics), antihypertensives, oral contraceptives, alcohol, opiates, and cocaine. Another important mechanism of GnRH inhibition is Cushing's disease from the elevation in serum glucocorticoids.

Holistic treatment of amenorrhea

While there exists a class of botanicals used in herbal medicine to induce menstruation called emmenagogues, the holistic treatment of amenorrhea rests on addressing the underlying factors, e.g. PCOD (see p. 57), autoimmunity, etc. An optimal body fat percentage should be encouraged through a healthy diet and moderate exercise. In Ayurveda, an absence of menstruation is a *vata* disorder, and thus general measures should be undertaken to regulate and balance the neuro-endocrine system, including the resolution of chronic emotional stress.

The treatment of hyperprolactinemia involves the usage of botanicals such as chasteberry (*Vitex agnus castus* seed) that has a dopaminergic activity, along with other herbs to support the hypothalamic-pituitary axis such as peony root (*Paeonia lactiflora* root), shu di huang (*Rehmannia glutinosa* prepared root), and licorice (*Glycyrrhiza glabra* root). The supplementation of zinc and vitamin B6 are useful as both are cofactors in dopamine synthesis and can be included in the treatment. It is important to weed through the various medications that could be causing this condition, as well as eliminating alcohol from the diet.

Where androgenization is part of the clinical pattern in amenorrhea, herbs to nurture and enhance the "feminine essence" should be included such as shatavari (*Asparagus racemosa* root) and peony (*Paeonia lactiflora* root), which increase the enzymatic conversion of testosterone to less potent androgens. Other herbs to competitively inhibit androgenic activity include damiana (*Turnera diffusa* herb), saw palmetto (*Serenoa serrulata* fruit), and sarsaparilla (*Smilax spp.* root). Phytoestrogens such as red clover blossoms (*Trifolium pratense* flower), true unicorn (*Aletris farinosa* root), and black cohosh (*Cimicifuga racemosa)* not only contribute to the estrogen pool but also increase the levels of SHBG that deactivate androgens.

Premenstrual syndrome

Premenstrual syndrome (PMS) refers to the different kinds of symptoms experienced by some women during the luteal and menstrual phase of the estrus cycle. It affects upwards of 75% of all women of menstruating age in varying degrees. The symptoms of PMS include both physical and psychological signs and symptoms. Physical symptoms of PMS include

- abdominal distension
- breast swelling and tenderness
- headaches
- changes in appetite
- food cravings
- fatigue
- dizziness
- weight gain
- fluid retention
- joint pain
- pelvic congestion
- poor immunity
- constipation
- diarrhea
- herpes outbreak
- acne

Psychological signs and symptoms of PMS include:

- insomnia
- poor memory
- grief, depression
- irritability
- anger
- anxiety
- poor concentration
- confusion

To a great extent many of the apparent "issues" associated with PMS are actually quite normal and natural as long as they aren't too severe, although a healthy menstrual cycle should provide minimal discomfort. The reality is that every 28 days or so a woman's body undergoes significant hormonal changes that have definite and measurable effects on both physiological and psychological function. While temperance is admirable quality in any person, it has long been associated as a particularly feminine virtue, suggesting that the emotional variability experienced by women as part of their normal experience is sometimes at odds with the needs of everyday society. This is reflected in the "red hut" traditions found all over the world, in

which menstruating women would gather and be to some extent sequestered, relieved of their regular duties, often ritualizing their shared experience through song, chanting, dance, and meditation.

As human societies all over the world have become more patriarchal, temperance was no longer seen as a virtue inasmuch as it was a necessity, and the natural emotional lability of women became stigmatized as a kind of pathology. In medieval Europe, physicians believed that this feminine "hysteria" arose as the result of a “wondering womb” that was searching the body looking for a baby. Likewise, the red hut tradition began to reflect not a woman's need to change up her routine during menses, but evidence of a stigma, in which a menstruating woman was seen as ritually impure or polluted in some way. Evidence of this stigma still remains to this day, from the backward chhaupati tradition followed by uneducated villagers in Nepal, to the modern medical profession in the West, which still views PMS as a kind of psychological disorder that must be treated with anti-depressants. Many companies have taken advantage of this taboo around menstruation to market their products, such as tampons and menstrual pads, selling the notion of women's liberation, while at the same time, reinforcing the notion that menstruation is something to be hidden.

The physical and psychological symptoms of PMS are in large part caused by fluctuations in neuroendocrinal agents including estrogen, progesterone, aldosterone, prolactin, dopamine, and endorphins:

- Estrogen: Elevated levels of estrogen relative to progesterone 5-10 days prior menses is thought to promote feelings of irritability and aggression by enhancing norepinepherine levels in the brain.
- Progesterone: A relative deficiency of progesterone 5-10 days prior menstruation allows for the elevation of aldosterone, enhancing sodium retention and the resultant edema. The progesterogenic effects of the luteal phase are also inhibited by elevated norepinepherine from emotional stress and elevated estrogen.
- Aldosterone: Aldosterone is a cause of premenstrual fluid retention, and is enhanced with stress, low progesterone, high estrogen, and a deficiency of magnesium.
- Prolactin: Women with PMS often have elevated levels and/or excessive sensitivity to prolactin, and if elevated during the luteal phase, promotes increased breast sensitivity and swelling.
- Dopamine: Dopamine is a prolactin antagonist, but is decreased under the influence of estrogen and a deficiency of magnesium and vitamin B_6. Dopamine also plays a role in regulating mood, and a deficiency is implicated in PMS-related anxiety, irritability, and emotional lability.
- Endorphins: Endorphins are natural opioids that elevate mood, and when decreased, can give rise to symptoms of depression. Endorphins also appear to regulate the secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) (Trickey 1998, 109-118).

Another much discussed factor in PMS is an eicosanoid imbalance, linked to the excessive consumption of n6 fatty acids, primarily in the form of seed oils or animal products that express a fatty acid imbalance (i.e. feedlot meat, farmed fish). The gut microbiome also appears

to have an important modulating influence on PMS, and under the influence of a relative estrogen excess can promote the growth of pathogens such as *Candida albicans*. The breast swelling and tenderness associated with elevated prolactin levels may be relieved by supplementation of vitamin B_6, which is a rate-limiting cofactor in the synthesis of dopamine. This same dopaminergic property can also be helpful to provide relief from depression and anxiety. Magnesium is another factor in dopamine synthesis, and thus in a similar fashion, a deficiency can lead to the depression and anxiety associated with PMS. Both vitamin B_6 and magnesium help to reduce vascular spasm and pain, and supplementation may help prevent and treat dysmenorrhea and cyclic breast pain (Trickey 1998, 109-118).

There are five different subcategories of PMS, a scheme first devised by G.E. Abraham, with each of these subtypes displaying a unique set of signs and symptoms. The following chart describes these subtypes and the mechanisms that contribute to their cause. It is important to note that a woman with PMS may experience more than one subtype, and/or may fluctuate between them.

Subgroup	Symptoms	Mechanisms
PMS A A = anxiety	anxiety nervousness mood swings nervous tension	estrogen excess progesterone deficiency liver congestion
PMS C C = craving	craving for sweets increased appetite palpitations fatigue dizziness headaches	hypoglycemia magnesium deficiency prostaglandin imbalance often occurs in association with PMS A
PMS D D = depression	depression poor memory grief confusion insomnia	estrogen deficiency
PMS H H = hydration	breast tenderness bloating weight gain edema	elevated aldosterone estrogen excess progesterone deficiency elevated prolactin
PMS P P = pain	lower back pain abdominal pain joint pain headaches	estrogen excess prostaglandin imbalance

(Trickey 1998, 118-121)

Holistic treatment of PMS A

The primary treatment of **PMS A** is to address the underlying mechanisms of hormonal balance while working to improve symptoms. A relative progesterone deficiency can be treated with chasteberry (*Vitex agnus castus* fruit), 40 gtt. of a 1:3 extract taken every morning for at least 6 months. Vitamin B_6, at a dose of between 100-600 mg daily, taken with 50-100 mg of a full spectrum B-complex, can be used 10-14 days prior menses to boost ovulation. In contrast, magnesium (as citrate, glycinate) can be taken at a dose of between 200-600 mg daily throughout the cycle. Cases that do not respond after at least three cycles of treatment can be

treated with natural progesterone cream, 20 mg applied on the forearm or inner thigh, once daily before bedtime, from mid-cycle (ovulation) to the first day of menstruation.

To address a relative estrogen excess, cholagogues are used to enhance the excretion of conjugated estrogen, including chai hu *(Bupleurum chinensis* root), barberry *(Berberis vulgaris* root), and dandelion *(Taraxacum officinalis* root). Phytoestrogenic herbs that compete with estrogen-binding sites may also be indicated, such as red clover *(Trifolium pratense* flower), as well as phytoestrogen-containing foods such as fermented and sprouted legumes. Fiber intake should be enhanced generally when trying to reduce estrogen levels, along with a reduction saturated fat and refined carbohydrate intake.

Other important measures in the treatment of PMS A include botanicals to reduce anxiety and pain, and promote a feeling of well-being and contentment. These include relaxing nervines include valerian *(Valeriana officinalis* root), skullcap *(Scutellaria lateriflora* herb), passionflower *(Passiflora incarnata* herb), and vervain *(Verbena officinalis* herb). Thymoleptics to improve mood include St. John's wort *(Hypericum perforatum* flower), cacao *(Theobroma cacao* seed), holy basil *(Ocimum sanctum* herb), damiana *(Turnera diffusa* herb), brahmi *(Bacopa monnieri* herb), pasqueflower *(Anenome occidentalis* herb), and ashwagandha *(Withania somnifera* root). Antispasmodic herbs to alleviate any pain or discomfort include wild yam *(Dioscorea villosa* tuber), crampbark *(Viburnum opulus* root bark), kava *(Piper methysticum* root bark), cannabis *(Cannabis indica* flower), and kratom (*Mitragyna speciosa* leaf). Adaptogens are also indicated in anxiety with exhaustion, including herbs ashwagandha *(Withania somnifera* root), shatavari *(Asparagus racemosa* root), dang gui (*Angelica sinensis* root), bai shao *(Paeonia lactiflora* root), and Siberian ginseng *(Eleuthrococcus senticosus* root).

Holistic treatment of PMS C

The primary treatment of PMC C is to regulate blood sugar levels, best accomplished by enhancing protein and fat intake, especially in the morning, while decreasing refined carbohydrate intake during the day. While smaller, more frequent meals can be helpful when the digestion is weak or if there is weight loss, most patients will benefit by reducing meal time frequency, as well as reducing the consumption of methylxanthine-containing beverages such as coffee and tea that promote blood sugar labilty. Supplementation with vitamin B_6 (100-300 mg daily, taken with a B-complex) along with magnesium citrate or glycinate (200-600 mg daily) may be useful to address hormonal imbalances. Chromium (250-500 mcg daily with meals) can be taken to improve insulin sensitivity.

Holistic treatment of PMS D

Different from the other types of PMS, the PMS D subtype that is associated with depression is related to a relative estrogen deficiency, and thus measures to enhance estrogen production or facilitate the cellular activities of estrogen are all helpful. Certain environmental contaminants such as lead, found in some fuels, paints, and other household products, can accumulate in the body and interfere with the activity of estrogen receptors. A diet that is very high in fiber can also lead to the excessive excretion of estrogen, and thus fiber intake should be carefully assessed in PMS D. Phytoestrogenic foods such as fermented legumes and sprouts can be included in the diet, along with phytoestrogenic herbs such as shatavari *(Asparagus racemosa* root), dang gui *(Angelica sinensis* root), red clover *(Trifolium pratense* flower), and black cohosh

(Cimicifuga racemosa root). Thymoleptic herbs to improve mood include St. John's wort *(Hypericum perforatum* flower), holy basil *(Ocimum sanctum* herb), damiana *(Turnera diffusa* herb), pasqueflower *(Anenome occidentalis* herb), and ashwagandha *(Withania somnifera* root). In particularly severe cases psychotropic herbs such as psilocybin mushrooms *(Psilocybe spp*.) can be taken a few times in a large doses as an entheogenic ritual (e.g. 5 g freshly dried mushrooms), or micro-dosed on a daily basis (150 mg bid) for a period of three months. Serotinergic foods that are rich in tryptophan (e.g. turkey and hard cheeses) can also be taken to enhance serotonin synthesis, or with severe depression, the biological precursor to serotonin, 5-HTP (100-300 mg daily).

Holistic treatment of PMS H

The treatment of PMS H is essentially the same as it is for PMS A, with the addition of treatments to correct aldosterone levels and sodium-potassium balance. Botanicals that are rich in potassium can be employed as an infusion, such as dandelion leaf *(Taraxacum officinalis* leaf), nettle *(Urtica dioica* herb), catnip *(Nepeta cataria* herb), and skullcap *(Scutellaria lateriflora* herb), along with potassium-rich foods such as kelp, raisins, avocados, apricots, potato skins, cantaloupe, and broccoli. Excessive salt consumption and herbs such as licorice *(Glycyrrhiza glabra* root) that promote sodium-retention should be avoided. Regular physical exercise to promote lymphatic flow and relieve edema is another important intervention in PMS H.

Holistic treatment of PMS P

PMS P relates to an increased sensitivity to pain, and in large part is related to an imbalance of the proinflammatory and pain-promoting eicosanoids, usually in conjunction with a relative estrogen excess. Dietary measures are taken to reduce n6 fatty acid consumption by avoiding seed oils and feed-lot/industrially-farmed animal produce, while increasing the consumption of n3 fatty acid sources including wild fish, leafy greens, sea vegetables, and certain seeds including hemp, flax, or chia. Magnesium and vitamin B_6 taken at dosages previously mentioned are also helpful to reduce pain sensitivity, along with general measures to reduce a relative estrogen excess, including an avoidance of refined carbohydrates and saturated fats.

Botanicals to inhibit the inflammatory cascade and pain sensitivity include those that exert antioxidant, antiinflammatory, and cholagogue effects, including turmeric *(Curcuma longa* rhizome), feverfew *(Tanacetum parthenium* herb), St. John's wort *(Hypericum perforatum* flower), chai hu *(Bupleurum falcatum* root), devil's claw *(Harpagophytum procumbens* root), and huang qin *(Scutellaria baicalensis* root). Herbs that have useful analgesic and anodyne properties include California poppy *(Eschscholzia californica* herb), wild lettuce root *(Lactuca virosa* root), Jamaican dogwood *(Piscidia erythrina* bark), white willow bark *(Salix alba* bark), pasqueflower *(Anenome occidentalis* herb), kava *(Piper methysticum* root bark), cannabis *(Cannabis indica* flower), and kratom (*Mitragyna speciosa* leaf).

Fibrocystic breast disease

Fibrocystic breast disease (FBD) is a common benign condition of premenopausal women that may or may not occur with the variance in hormonal levels typically experienced during the estrus cycle. Although many women display areas of relatively indistinct breast

